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# FINAL BASELINE RISK ASSESSMENT

JUNE 1991

HAVERTOWN PCP (RI/FS) SITE

HAVERFORD TOWNSHIP, PENNSYLVANIA

TETRA TECH, INC.

TCN 4212

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# TABLE OF CONTENTS

TABLE OF CONTENTS
(continued)

LIST OF TABLES:

## 6.0 BASELINE RISK ASSESSMENT

The baseline risk assessment report for the Havertown PCP site quantifies potential human health risks and environmental impacts associated with the site. The RI baseline risk assessment determines whether the chemicals of potential concern at the Havertown PCP site pose a current or future risk to human health and the environment under the no-action alternative (i.e., in the absence of remediation of the site). According to the NCP (EPA 1990a), the baseline risk assessment "...provides a basis for determining whether remedial action is necessary and the justification for performing remedial actions." The baseline risk assessment was prepared in keeping with available Federal EPA guidance for conducting Superfund risk assessments, including Risk Assessment Guidance for Superfund (EPA 1990b, 1989a,b). In addition, the baseline risk assessment was prepared using EPA Region III specific guidance (EPA 1991a).

The baseline risk assessment consists of two assessments: human health assessment and ecological assessment. The evaluation of the potential noncarcinogenic and carcinogenic human health risks from exposure to chemicals released from the site is presented in Section 6.1. The evaluation of the potential terrestrial and aquatic ecological impacts due to chemical releases from the site is presented in Section 6.2.

## 6.1 HUMAN HEALTH ASSESSMENT

### 6.1.1 Introduction to the Human Health Assessment

The human health assessment for the Havertown PCP site quantifies potential human health risks associated with the site. The human health risk assessment process consists of four basic steps which form the outline of this report.

STEP 1. Selection of Chemicals of Potential Concern - (Section 6.1.2) Monitoring data collected as part of the Remedial Investigation (RI) are analyzed and chemicals of potential concern are selected. Of the chemicals detected at the site, chemicals of potential concern are selected based on an evaluation of risk factors (which quantify the relative percent contribution of risk); frequency of detection; low toxicity to humans (i.e., essential human nutrient); and background concentrations. Selected chemicals of potential concern are evaluated further in the report.

STEP 2. Exposure Assessment - (Section 6.1.3) Exposure pathways are identified based on an evaluation of the environmental setting of the site and the environmental fate and transport of chemicals of potential concern. Exposure pathways are selected for both current and future land-use of the site. Exposure point concentrations and exposures are estimated for each chemical of potential concern for the exposure pathways quantitatively evaluated in this report.

STEP 3. Toxicity Assessment - (Section 6.1.4) Toxicity criteria for assessing carcinogenic and noncarcinogenic risks for the selected chemicals of potential concern are presented and evaluated.

STEP 4. Risk Characterization - (Section 6.1.5) The exposure estimates presented in Section 6.1.3 and the toxicity criteria presented in

Section 6.1.4 are combined to estimate potential carcinogenic and noncarcinogenic risks for the exposure pathways quantitatively evaluated in this report. These risks characterize the potential human health impact associated with the Havertown PCP site.

In addition, the uncertainties associated with the human health risk assessment process and the conclusions of the report are presented in Section 6.1.6 and Section 6.1.7, respectively.

### 6.1.2 Selection of Chemicals of Potential Concern

This section selects chemicals of potential concern that will be evaluated further in the human health risk assessment for the Havertown PCP site. Chemicals of potential concern will be selected for groundwater, surface water and sediment from Naylors Run, and storm sewer surface water and sediment. As discussed in Section 1, soils at the Havertown PCP site were evaluated as a separate operable unit under a previous RI/FS effort. As part of this RI, potential risks associated with the surface and subsurface soils from the Havertown PCP site were evaluated in the "Havertown PCP Site Risk Assessment", prepared by Greeley-Polhemus Group, Inc (1989). Thus, potential exposure and risks from chemicals present in surface and subsurface soil will not be reevaluated in this report. The Havertown PCP Site Risk Assessment (Greeley-Polhemus Group, 1989) did not evaluate the potential risks associated with use of groundwater at the site. In addition, exposure estimates for surface water and sediments from Naylors Run were based on limited monitoring data and certain exposure parameter values were well below standard "reasonable maximum" values (EPA 1989a). Therefore, the human health risks associated with groundwater (under future land-use conditions) and surface water and sediments (under current land-use conditions) will be evaluated in this report using current risk assessment methodologies (EPA 1989a).

The methods used to analyze monitoring data and select chemicals of potential concern for the Havertown PCP site are presented in Section 6.1.2.1 and Section 6.1.2.2, respectively. Chemicals of potential concern selected for groundwater, surface water, and sediment are presented in Sections 6.1.2.3, 6.1.2.4, and 6.1.2.5, respectively. A summary of chemicals of potential concern selected for all media is presented in Section 6.1.2.6.

6.1.2.1 Methods for Evaluating and Analyzing Data

A significant quantity of data were collected from the Havertown PCP site. Monitoring results were collected from groundwater, surface water and sediment from Naylors Run, and storm sewer sediment and surface water. The RI monitoring data were analyzed using several screening procedures, in order to derive a database suitable for risk assessment purposes (EPA 1989a). The chemical data presented in Appendices B and D of the RI were modified according to the screening steps outlined in this section in order to derive a suitable database. Thus, differences between the data presented in Section 6 and other portions of the RI are reflective of the modifications in the database which must be made for performing the human health risk assessment. Factors considered when evaluating the RI monitoring data included potential blank contamination, QA/QC procedures and codes, high detection limits, combining split and duplicate samples, and summing chemical mixtures. The screening procedures used to analyze chemical concentration data collected for the Havertown PCP site are discussed below.

- Pursuant to EPA (1989a) guidance, common laboratory contaminants (e.g., acetone, 2-butanone, methylene chloride, phthalates, and toluene) detected in on-site samples which were within ten times the concentration detected in field or trip blank samples were not included in the analysis. This screening method is used because chemicals detected in blank samples and on-site samples may not be actually present in the media sampled. Likewise, uncommon laboratory contaminants (i.e., chemicals not considered above) detected in on-site samples which were within five times the concentration detected in field or trip blank samples were not included in the analysis (EPA 1989a). These chemicals were flagged with a "B" qualifier by the data validator and were deleted from the RI monitoring database. One particular chemical of potential concern deleted from the groundwater database due to field and/or lab contamination was lead. The levels of lead detected in

monitoring well samples, however, were well below the Federal Maximum Contamination Level (MCLs) for lead of 50 ug/L.

- Monitoring data qualified "unreliable" with a "R" was based on data validation procedures were deleted from the RI monitoring database.

- Detection limits (DL) that exceeded two times the maximum detected concentration of a chemical were not included when estimating mean concentrations for the site, but were included when estimating the frequency of detection. For example, if a chemical was not detected in one sample and the DL was 100 ug/L and the maximum detected concentration at the site was 10 ug/L, then the DL was not included when calculating various statistics since the DL would bias the results.

- One-half the reported DL was used as the concentration for monitoring data qualified with an "U" or "UJ" (i.e., a non-detect).

- Chemicals that were never detected in a given media were deleted from the RI monitoring database.

- Laboratory variance tends to be normally distributed; therefore, the arithmetic mean (and not the geometric mean) was used to combine the split and duplicate samples. If a chemical was not detected in one sample but detected in the split sample, then the chemical was considered to be detected in the combined sample for the purpose of calculating frequency of detection.

- For certain chemical groups, toxicity criteria were only available for certain chemical constituents from the chemical group. Thus, the concentrations of chemical constituents from the following chemical classes were summed for each sample:

- alpha chlordanes and gamma chlordanes;
- endosulfans I and endosulfans II;
- DDT, DDE, and DDD;
- polychlorinated biphenyls;
- dioxin and furans; and
- carcinogenic polycyclic aromatic hydrocarbons (PAHs).

The total concentration for the above mentioned chemical classes were calculated using an unweighted sum, with the exception of the carcinogenic PAHs, dioxins, and furans. Thus, the toxicity of each chemical in the chemical class were assumed to have the same potency (with the exception of PAHs, dioxins and furans). The total concentration of carcinogenic PAHs for each sample was calculated using a weighted sum by applying toxicity equivalency factors (TEFs) (Clement 1988). TEFs quantify the cancer potency of carcinogenic PAHs relative to benzo(a)pyrene. For each sample, TEFs were multiplied by the chemical concentration and then summed to derive the concentration of benzo(a)pyrene (Equivalent). Available TEFs for carcinogenic PAHs are presented in Table 6-1. Essentially, the same approach was used to sum dioxin and furan congeners to estimate the 2,3,7,8-TCDD (Equivalent) concentration for each sample. TEFs used to estimate 2,3,7,8-TCDD (Equivalent) concentrations are presented in Table 6-2.

- Various summary statistics were calculated for each chemical

Table 6-1

Relative Toxicity Equivalency Factors (TEFs)
Derived for Carcinogenic PAHs (a)

| Carcinogenic PAH | TEF |
|---|---|
| Anthanthrene | 0.320 (b) |
| Benzo(a)pyrene | 1.0 |
| Benzo(e)pyrene | 0.004 (b) |
| Benzo(a)anthracene | 0.145 (c) |
| Benzo(b)fluoranthene | 0.140 (b) |
| Benzo(j)fluoranthene | 0.061 (d) |
| Benzo(k)fluoranthene | 0.066 (b) |
| Benzo(g,h,i)perylene | 0.022 (b) |
| Chrysene | 0.0044 (e) |
| Cyclopetadieno(c,d)pyrene | 0.023 (d) |
| Dibenz(a,h)anthracene | 1.11 (e) |
| Indeno (1,2,3-c,d) pyrene | 0.232 (b) |
| Pyrene | 0.081 (f) |

(a) Adopted from ICF - Clement (1988).
(b) Deutsch-Wenzel et al. (1983).
(c) Bingham and Falk (1969).
(d) Habs et al. (1980).
(e) Wynder and Hoffmann (1959).
(f) Wislocki et al. (1986).

Table 6-2

Relative Toxicity Equivalency Factors (TEFs)
Derived for 2,3,7,8-TCDD(a)

| Isomer | TEF (a) |
|---|---|
| 2378-TCDD | 1 |
| Other TCDD | 0 |
| 12378-PeCDD | 0.5 |
| Other PeCDD | 0 |
| 123478-HxCDD | 0.1 |
| 123678-HxCDD | 0.1 |
| 123789-HxCDD | 0.1 |
| Other HxCDD | 0 |
| 1234678-HpCDD | 0.01 |
| Other HpCDD | 0 |
| OCDD | 0.001 |
| 12378-TCDF | 0.1 |
| Other TCDF | 0 |
| 12378-PeCDF | 0.05 |
| 23478-PeCDF | 0.5 |
| Other PeCDF | 0 |
| 123478-HxCDF | 0.1 |
| 123678-HxCDF | 0.1 |
| 234678-HxCDF | 0.1 |
| 123789-HxCDF | 0.1 |
| Other HxCDF | 0 |
| 1234678-HpCDF | 0.01 |
| 1234789-HpCDF | 0.01 |
| Other HpCDF | 0 |
| OCDF | 0.001 |

(a) International Toxicity Equivalency Factors 1985.

Table 6-3

Summary of Chemicals Detected in
Groundwater at the Havertown PCP Site

| Compound | SF Risk Factor (a) | RfD Risk Factor (b) | Human Nutrient (c) | Concentration Data (Units: ug/L)(d) Frequency of Detection | Minimum Detected | Geometric Mean | Maximum Detected |
|---|---|---|---|---|---|---|---|
| Organics: | | | | | | | |
| Acetone | -- | <1% | No | 1/21 | 4.0 | 7.9 | 4.0 |
| •Benzene | <1% | -- | No | 13/28 | 1.0 | 11.0 | 270.0 |
| 2-Butanone | -- | <1% | No | 1/28 | 3.0 | NC | 3.0 |
| Carbon Disulfide | -- | <1% | No | 1/28 | 3.0 | 2.5 | 3.0 |
| Chloroethane | <1% | -- | No | 1/28 | 5.0 | NC | 5.0 |
| Di-n-butylphthalate | -- | <1% | No | 2/24 | .5 | NC | .9 |
| Di-n-octylphthalate | -- | -- | No | 1/26 | .3 | NC | .3 |
| •Dibenzofuran | -- | -- | No | 7/27 | 1.0 | 30.0 | 1,300.0 |
| •1,2-Dichloroethene (total) | -- | <1% | No | 12/28 | 1.0 | 15.0 | 270.0 |
| Dieldrin | <1% | <1% | No | 1/28 | .6 | .2 | .6 |
| Endosulfan II | -- | <1% | No | 1/28 | 3.5 | .2 | 3.5 |
| Ethyl Benzene | -- | <1% | No | 1/28 | 160.0 | 6.0 | 160.0 |
| •bis(2-Ethylhexyl)phthalate | <1% | <1% | No | 2/17 | 37.5 | 39.0 | 100.5 |
| 4-Methyl-2-pentanone | -- | -- | No | 1/28 | 2.0 | NC | 2.0 |
| Methylene Chloride | <1% | <1% | No | 4/21 | 14.5 | 9.9 | 590.0 |
| Polycyclic Aromatic Hydrocarbons | | | | | | | |
| •2-Methylnaphthalene | -- | -- | No | 18/28 | 7.0 | 59.0 | 21,000.0 |
| •Acenaphthene | -- | -- | No | 7/27 | .8 | 32.0 | 1,700.0 |
| •Acenaphthylene | -- | -- | No | 3/26 | 2.0 | 6.7 | 16.0 |
| •Anthracene | -- | -- | No | 6/27 | .4 | 33.0 | 1,900.0 |
| •Benzo(a)anthracene | -- | -- | No | 2/27 | 25.0 | 17.0 | 190.0 |
| •Benzo(a)pyrene (Equivalent)(e) | 20.0% | -- | No | 7/27 | .9 | 19.0 | 741.9 |
| •Chrysene | -- | -- | No | 1/27 | 240.0 | 30.0 | 240.0 |
| •Fluoranthene | -- | <1% | No | 3/27 | .3 | 32.0 | 810.0 |
| Fluorene | -- | <1% | No | 8/28 | 1.0 | 38.0 | 2,900.0 |
| •Naphthalene | -- | 2.4% | No | 18/28 | 4.0 | 85.0 | 24,000.0 |
| •Phenanthrene | -- | -- | No | 12/28 | .7 | 40.0 | 12,000.0 |
| •Pyrene | -- | -- | No | 5/27 | .5 | 35.0 | 1,300.0 |
| •Pentachlorophenol | 21.6% | 1% | No | 24/28 | 14.0 | 670.0 | 80,000.0 |
| •2,3,7,8-TCDD (Equivalent)(f) | 58.4% | 96.3% | No | 23/28 | 0.001ppt | 0.17ppt | 173.7ppt |
| Toluene | -- | <1% | No | 3/28 | 2.0 | 5.6 | 92.0 |
| •Trichloroethene | <1% | <1% | No | 13/28 | 1.0 | 17.0 | 630.0 |
| •Vinyl Chloride | <1% | -- | No | 5/28 | 3.0 | 6.3 | 16.5 |
| Xylenes (total) | -- | <1% | No | 17/28 | 2.0 | 34.0 | 1,700.0 |
| Inorganics: | | | | | | | |
| •Aluminum | -- | -- | No | 12/27 | 31.4 | 30.0 | 2,390.0 |
| •Arsenic | <1% | <1% | No | 11/28 | 2.0 | 2.2 | 28.0 |
| Barium | -- | <1% | No | 28/28 | 21.0 | 69.0 | 357.0 |
| Cadmium | -- | <1% | No | 1/28 | 3.4 | 1.5 | 3.4 |
| Calcium | -- | -- | Yes | 28/28 | 13,400.0 | 32,000.0 | 110,000.0 |
| Chromium | -- | <1% | No | 1/28 | 21.6 | 3.2 | 21.6 |
| •Cobalt | -- | -- | No | 26/28 | 6.4 | 39.0 | 413.0 |
| Copper | -- | <1% | Yes | 3/28 | 3.9 | 1.8 | 13.6 |
| Iron | -- | -- | Yes | 27/28 | 31.7 | 2,500.0 | 31,400.0 |
| Magnesium | -- | -- | Yes | 28/28 | 7,980.0 | 15,000.0 | 81,600.0 |
| •Manganese | -- | <1% | No | 28/28 | 28.5 | 5,200.0 | 22,600.0 |
| Nickel | -- | <1% | No | 9/28 | 6.5 | 5.3 | 64.7 |
| Potassium | -- | -- | Yes | 28/28 | 1,390.0 | 5,700.0 | 22,000.0 |
| Sodium | -- | -- | Yes | 28/28 | 9,190.0 | 27,000.0 | 137,000.0 |
| Thallium | -- | <1% | No | 1/28 | 4.2 | 1.3 | 4.2 |
| Vanadium | -- | <1% | No | 3/28 | 2.8 | 1.7 | 19.8 |
| Zinc | -- | <1% | Yes | 7/7 | 33.2 | 100.0 | 243.0 |

NC not calculated
• Chemicals of potential concern
-- No toxicity criteria
(a) Percent contribution of carcinogenic risk based on the exposure point concentration and the slope factor (see text for further discussion).
(b) Percent contribution of non-carcinogenic risk based on the exposure point concentration and the RfD (see text for further discussion).
(c) Compound is an essential human nutrient. Concentrations of compound would result in exposures that are less than the Recommended Daily Allowance (RDA).
(d) Data analyzed according to data screening procedures outlined in Section 6.1.2.1. Frequency of detection is the number of detected concentrations divided by the number of samples (which may vary due to blank related contamination). Minimum and maximum concentration may be the average of duplicate samples.
(e) Concentrations of anthracene, benzo(a)anthracene, chrysene, and pyrene were summed using Toxicity Equivalency Factors (TEFs) to calculate total benzo(a)pyrene equivalents.
(f) 2,3,7,8-TCDD (Equivalent) calculated by summing dioxin and furan congener data using 2,3,7,8-TCDD TEFs.

Table 6-4

Tentatively Identified Compounds (TICs)
Detected in Groundwater at the Havertown PCP Site

| TIC | Range of Concentrations (ug/L) |
|---|---|
| Alkylbenzene | 4-3,600 |
| Benzofuran | 4-6 |
| 1,2-Dimethylnapnthalene | 4.2-77 |
| 1,3-Dimethylnaphthalene | 3.2-15 |
| 1,4-Dimethylnaphthalene | 46 |
| 1,5-Dimethylnapnthalene | 3-57 |
| 1,7-Dimethylnapnthalene | 4.3 |
| 1,8-Dimethylnaphthalene | 7-51 |
| 2,3-Dimethylnapnthalene | 3 |
| 2,5-Dimethylphenanthrene | 19-30 |
| Ethenylmethylbenzene | 34 |
| 2-Ethyl-1,1-biphenyl | 26 |
| 1-Ethylidenenaphthalene | 21 |
| 1-Ethyl-2-methylbenzene | 13-160 |
| 1-Ethyl-4-methylbenzene | 88 |
| 2-Ethylnaphthalene | 29 |
| Hexadecanoic acid | 6 |
| 1-Methylanthracene | 42 |
| 2-Methylanthracene | 27 |
| Methylcyclopentane | 76 |
| 9-Methyl-9H-fluorene | 26 |
| 1-Methylnapnthalene | 5.2-15 |
| 3-Methylphenanthrene | 29 |
| 2,3,5,6-Tetrachlorophenol | 3-6 |
| Tetramethylbenzene | 14-33 |
| 1,2,3-Trimethylbenzene | 8-40 |
| 1,2,4-Trimethylbenzene | 12 |
| 1,3,5-Trimethylbenzene | 110 |
| 1,4,5-Trimethylnapnthalene | 3-47 |
| 1,4,6-Trimethylnaphthalene | 1-32 |
| 1,6,7-Trimethylnaphthalene | 26 |
| 2,3,6-Trimethylnaphthalene | 26 |
| Unknown compound | 6-1000 |
| Unknown hydrocarbon | 6-1600 |
| Unknown PNA | 17 |

detected in a given media including: frequency of detection, geometric means, and range of detected concentrations. Most chemical distributions in nature tend to be lognormally distributed except for abundant metals such as aluminum and iron (Connor and Shacklette 1975, Dean 1981, Esmen and Hammad 1977, and Ott 1988). Theoretically, the geometric mean represents the median (i.e., 50 percentile) of the chemical distribution. Other statistics from the chemical distribution were used to estimate exposure point concentrations for the purpose of estimating exposure. The methods used to estimate these statistics (e.g., the 95th upper confidence limit on the arithmetic mean) are presented in Section 6.1.3.2.

6.1.2.2 Methods for Selecting Chemicals of Potential Concern

Only a subset of the chemicals detected at the site were selected as chemicals of potential concern for further evaluation in this report. Generally, chemicals of potential concern are selected based on an evaluation of background concentrations; risk factors which quantify the relative percent contribution of risk; low human toxicity (i.e., essential human nutrients); and to some extent frequency of detection. In addition, tentatively identified compounds were not selected as chemicals of potential concern and; thus, were not quantitatively evaluated in the report (these chemicals were qualitatively evaluated, however). In order to be conservative, chemicals which are not essential human nutrients and appear to be elevated above background levels, but do not have available toxicity criteria, were selected as chemicals of potential concern. The uncertainty associated with not being able to quantitatively evaluate these chemicals in the risk assessment will be discussed in the sections to follow.

The methods used to select chemicals of potential concern for the Havertown PCP site are discussed below.

Background Comparison - Comparing chemical concentrations detected at the site with background concentrations is important in order to properly delineate whether certain chemicals of concern are associated with site activities or from

natural background. The presence of certain inorganic chemicals detected at the site may be due to natural background, while certain organic compounds such as PAHs may be due to anthropomorphic activities (e.g., incomplete combustion of alkanes in automobiles may form PAHs). For the Havertown PCP site, however, site-specific background concentrations were not available.

For groundwater, monitoring wells located slightly upgradient from the site had significant organic contamination which is characteristic of the Havertown PCP site. No additional upgradient wells were identified for sampling purposes during the RI. In addition, monitoring wells downgradient from the site also had significant contamination which showed that they may be influenced by contaminant releases from the site. Thus, no groundwater background samples were available. Lack of site-specific background samples for this site, however, does not impact the results or conclusion of the baseline risk assessment because inorganic compounds did not significantly contribute to carcinogenic risk nor noncarcinogenic risk, as discussed in sections to follow. Unlike the inorganic compounds, PAHs did significantly contribute to the risks presented in this report. Although PAHs may be present at some sites due to anthropogenic causes, the disposal history of the Havertown PCP site indicates that it is the source of PAH contamination in the area.

For surface water and sediment, the Havertown PCP site is located at the headwaters of Naylors Run. Stations located upstream of the catch basin may be influenced from surface water runoff. In fact, the highest detected levels of certain carcinogenic PAHs were found upstream of the catch basin. Thus, no upstream locations were available for sampling. Unlike groundwater, inorganic compounds such as arsenic, chromium, manganese, and thallium did significantly contribute to the risks associated with sediments in Naylors Run, as discussed in the sections to follow. Due to the lack of site-specific background, it is uncertain to what extent the site contributed to these compounds found in Naylors Run.

Risk Factors - Of those chemicals considered to be elevated above background, only those which may significantly contribute to carcinogenic and noncarcinogenic risks were selected for further evaluation in this report. Chemicals which would significantly contribute to estimated risk were identified by calculating the percent contribution of carcinogenic risk and noncarcinogenic risk (EPA 1989a). Chemicals which contributed greater than 1 percent of the total carcinogenic risk or noncarcinogenic risk were selected as chemicals of potential concern. This method can be used for any exposure pathway, since the same exposure parameters would be applied to all chemicals.[1] As previously discussed, detected chemicals without available toxicity criteria were selected as chemicals of potential concern in order to be conservative. Chemicals detected in groundwater with toxicity criteria which contributed less than 1 percent of the total risk, but exceeded available Applicable or Relevant and Appropriate Requirements (ARARs) for drinking water (e.g., Federal Maximum Contamination Levels [MCLs]) also were selected as chemicals of potential concern.

Slope factors and reference doses (RfDs) used to calculate risk factors were obtained from the Integrated Risk Information System (IRIS) (EPA 1991b) and the 4th Quarter Health Effects Assessment Summary Tables (HEAST) (EPA 1990c). These sources are discussed further in Section 6.1.4 of this report.

The percent contribution of carcinogenic risk for each detected chemical was calculated using the following equation:

$$\%CCR_i = \frac{EPC_i * SF_i}{\sum_{j=1}^{n} EPC_j * SF_j} * 100$$

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1 The only exception to this rule is when the exposure estimate is dependent on the physicochemical properties of each chemical (e.g., dermal permeability).

$\%CCR_i$ = Percent contribution of carcinogenic risk for $chemical_i$;
$EPC_i$ = Exposure Point Concentration for $chemical_i$ (see Section 6.1.3.3 for discussion of the derivation of exposure point concentrations); and
$SF_i$ = Slope Factor for $chemical_i$.

The denominator of the equation sums the risk scores (i.e., exposure point concentration for $chemical_j$ multiplied by the slope factor for $chemical_j$) for all chemicals with available toxicity criteria.

The percent contribution of noncarcinogenic risk for each detected chemical was calculated using the following equation:

$$\%CNR_i = \frac{EPC_i/RfD_i}{\sum_{j=1}^{n} EPC_j/RfD_j} * 100$$

where:

$\%CNR_i$ = Percent contribution of noncarcinogenic risk for $chemical_i$;
$EPC_i$ = Exposure Point Concentration for $chemical_i$ (see Section 6.1.3.3 for discussion of the derivation of exposure point concentrations); and
$RfD_i$ = Reference dose for $chemical_i$.

The denominator of the equation sums the noncarcinogenic risk scores (i.e., exposure point concentration for $chemical_j$ divided by the RfD for $chemical_j$) for all chemicals with available toxicity criteria.

- As recommended in EPA (1989a) guidance, tentatively identified compounds

(TICs) were not selected as chemicals of potential concern for quantitative evaluation, rather TICs were evaluated qualitatively in this report.

- Inorganic compounds considered essential human macronutrients (i.e., calcium, iron, magnesium, potassium, and sodium) have low toxicity to humans and thus were not selected as chemicals of potential concern. Micronutrient inorganics such as copper and zinc have slightly higher toxicity than do the macronutrient compounds and were evaluated in a similar manner to other chemicals detected at the site.

- Certain chemicals that are detected infrequently (i.e., less than 5 percent) at concentrations below the detection limit also were not selected as chemicals of potential concern. However, if the chemical significantly contributed to risk or the maximum concentration exceeded ARARs, then the chemical was selected as a chemical of potential concern.

Chemicals of potential concern selected for groundwater, surface water, and sediment from Naylors Run and the storm sewers are presented in the following sections.

6.1.2.3 Groundwater

Groundwater samples were collected from 28 monitoring wells of which 18 were from 6 cluster wells (a well cluster consists of 3 wells installed in the shallow, intermediate, and deep zones of the aquifer). Eleven wells are located on the National Wood Preservers (NWP) site, while the remaining wells are located on the Philadelphia Chewing Gum (PCG) site or further downgradient of the site near Naylors Run. These wells are installed in different zones of the same aquifer system and there is little difference in elevation between the screening levels of these wells. All of the monitoring wells were analyzed collectively for the purpose of selecting chemicals of potential concern. However, only wells

installed in the saprolite and deep bedrock of the aquifer were evaluated for the purpose of estimating exposure and risk. As previously discussed, wells hydrogeologically upgradient from the NWP site still had significant levels of organic contamination; therefore, these wells could not be used for evaluating site-specific background for groundwater. Only monitoring data from sampling Round 2 were validated for use in the risk assessment. Monitoring data from Round 1 sampling were used for screening purposes only. In general, monitoring data from Round 1 sampling was similar to levels found in Round 2. Further movement of the plume was evident by increased detection of chemicals of potential concern in the farthest downgradient well locations (see Section 4 of this report for further discussion on Round 1 versus Round 2 sampling).

Table 6-3 presents chemicals detected in groundwater monitoring wells at the Havertown PCP site. Chemicals of potential concern identified in Table 6-3 were selected based on the criteria presented in Section 6.1.2.2. The most commonly detected organic chemicals in groundwater included pentachlorophenol (PCP) and PAHs. Of the chemicals detected with available toxicity criteria, benzo(a)pyrene (Equivalent), PCP, and 2,3,7,8-TCDD (Equivalent) contributed to more than 99 percent of the relative cancer risk. Naphthalene and 2,3,7,8-TCDD (Equivalent) contributed 99 percent of the relative noncancer risk. The highest detected concentrations for these organic chemicals were found in monitoring wells directly downgradient of the NWP site (HAV-02, HAV-04, and R-2). Inorganic chemicals did not appear to contribute significantly to the potential carcinogenic and noncarcinogenic risks. This may indicate that the lack of background data for groundwater may not contribute significantly to the overall conclusions of the groundwater risk assessment. TICs detected in groundwater samples are presented in Table 6-4. The majority of the TICs consisted of alkyl benzene compounds and PAHs, particularly alkyl naphthalene. The presence of these chemicals is consistent with the disposal history of the site.

6.1.2.4 Surface Water

During the first round of sampling, surface water samples were collected from five locations in Naylors Run downstream from NWP and three locations in storm sewers leading to Naylors Run (see Section 4 for further discussion of sampling locations). No samples could be collected upstream of the NWP site given that the site is located at the headwaters of Naylors Run. Thus, there were no background samples to determine water quality independent of possible site contamination. Overall, higher contaminant concentrations were measured in the storm sewer than in Naylor Run. The decrease in chemical concentrations may be due to dilution or volatization.

The second round of surface water data was collected primarily to fill data gaps for conducting the aquatic ecological assessment. These data were incorporated into the ecological assessment. In general, the levels of PCP and dioxin were significantly lower further downstream.

Naylors Run Surface Water - Table 6-5 presents chemicals detected in surface water from Naylors Run. Chemicals of potential concern identified in Table 6-5 were selected based on the criteria presented in Section 6.1.2.2. With the exception of PCP, most samples contained concentrations of organics at or near the detection limit. As shown in Table 6-5, PCP and 2,3,7,8-TCDD (Equivalent) are the principal carcinogens of concern in surface water (contributed over 90 percent of the relative carcinogenic risk). Other compounds which had significant risk factors for this media include heptachlor epoxide, dieldrin, and benzo(a)pyrene (Equivalent). 2,3,7,8-TCDD (Equivalent) contributed over 90 percent of the relative noncarcinogenic risk associated with surface water. The highest detected concentrations of PCP and 2,3,7,8-TCDD were found in the sample collected from the catch basin (NAY-AQ-03). The inorganic chemicals detected in surface water are generally not considered to be carcinogenic; therefore, the relative carcinogenic risks for these chemicals were not be calculated. Levels of manganese and thallium in surface water indicate that these inorganics may be significant chemicals of potential concern.

Table 6-5

Summary of Chemicals Detected in
Naylors Run Surface Water at the Havertown PCP Site

| Compound | SF Risk Factor (a) | RfD Risk Factor (b) | Human Nutrient (c) | Concentration Data (Units: ug/L)(d) Frequency of Detection | Minimum Detected | Geometric Mean | Maximum Detected |
|---|---|---|---|---|---|---|---|
| Organics: | | | | | | | |
| Acetone | -- | <1% | No | 1/1 | 7.0 | NC | 7.0 |
| gamma-BHC | <1% | -- | No | 1/5 | .1 | .0 | .1 |
| Benzene | <1% | -- | No | 3/5 | 10.5 | 8.9 | 31.0 |
| 4,4'-DDD | <1% | <1% | No | 2/5 | .2 | .1 | .4 |
| 1,2-Dichloroethene (total) | -- | <1% | No | 3/5 | 2.3 | 2.6 | 3.0 |
| •Dieldrin | 2.7% | <1% | No | 2/5 | .1 | .1 | .3 |
| Ethyl Benzene | -- | <1% | No | 3/5 | 6.0 | 8.0 | 33.0 |
| bis(2-Ethylhexyl)phthalate | <1% | <1% | No | 1/1 | 4.0 | NC | 4.0 |
| •Heptachlor Epoxide | 3.4% | 1.3% | No | 1/5 | .8 | .1 | .8 |
| Methylene Chloride | <1% | <1% | No | 1/1 | 13.0 | NC | 13.0 |
| •Pyrene | -- | -- | No | 1/5 | 4.0 | NC | 4.0 |
| •Benzo(a)pyrene (Equivalent)(e) | 1.8% | -- | No | 1/5 | .3 | NC | .3 |
| Fluorene | -- | <1% | No | 1/5 | 2.0 | NC | 2.0 |
| •Pentachlorophenol | 69.8% | 1.2% | No | 5/5 | 18.0 | 160.0 | 1,200.0 |
| •2,3,7,8-TCDD (Equivalent)(f) | 21.8% | 92.0% | No | 2/5 | 0.002ppt | 0.23ppt | 0.30ppt |
| Toluene | -- | <1% | No | 1/5 | 3.0 | 2.6 | 3.0 |
| Trichloroethene | <1% | <1% | No | 2/5 | 7.0 | 3.8 | 7.0 |
| Xylenes (total) | -- | <1% | No | 3/5 | 44.0 | 23.0 | [redacted]0 |
| Inorganics: | | | | | | | |
| •Aluminum | -- | -- | No | 3/5 | 53.5 | 43.0 | 147.0 |
| Barium | -- | <1% | No | 5/5 | 25.9 | 65.0 | 87.5 |
| Calcium | -- | -- | Yes | 5/5 | 15,700.0 | 22,000.0 | 28,400.0 |
| Chromium | -- | <1% | No | 1/5 | 4.2 | 1.8 | 4.2 |
| •Cobalt | -- | -- | No | 3/5 | 29.8 | 14.0 | 37.8 |
| Iron | -- | -- | Yes | 4/4 | 828.0 | 3,500.0 | 7,920.0 |
| •Lead | -- | -- | No | 5/5 | 2.3 | 5.5 | 12.9 |
| Magnesium | -- | -- | Yes | 5/5 | 3,570.0 | 9,400.0 | 14,000.0 |
| •Manganese | -- | 3.1% | No | 5/5 | 228.0 | 2,500.0 | 10,100.0 |
| Potassium | -- | -- | Yes | 5/5 | 3,780.0 | 4,600.0 | 5,070.0 |
| Silver | -- | <1% | No | 1/5 | 3.2 | 2.2 | 3.2 |
| Sodium | -- | -- | Yes | 5/5 | 13,700.0 | 21,000.0 | 30,500.0 |
| •Thallium | -- | 1.5% | No | 3/5 | 2.2 | 1.9 | 3.3 |
| Zinc | -- | <1% | Yes | 2/2 | 41.2 | NC | 74.1 |

NC Not calculated
• Chemicals of potential concern
-- No toxicity criteria
(a) Percent contribution of carcinogenic risk based on the exposure point concentration and the slope factor (see text for further discussion).
(b) Percent contribution of non-carcinogenic risk based on the exposure point concentration and the RfD (see text for further discussion).
(c) Compound is an essential human nutrient. Concentrations of compound would result in exposures that are less than the Recommended Daily Allowance (RDA).
(d) Data analyzed according to data screening procedures outlined in Section 6.1.2.1. Frequency of detection is the number of detected concentrations divided by the number of samples (which may vary due to blank related contamination). Minimun and maximum concentration may be the average of duplicate samples.
(e) Concentration of pyrene multiplied by Toxicity Equivalency Factor (TEF) to estimate benzo(a)pyrene (Equivalent).
(f) 2,3,7,8-TCDD (Equivalent) calculated by summing dioxin and furan congener data using 2,3,7,8-TCDD TEFS.

Storm Sewer Surface Water - Table 6-6 presents chemicals detected in surface water from storm sewers. Chemicals of potential concern identified in Table 6-6 were selected based on the criteria presented in Section 6.1.2.2. Most organic chemicals were detected in the storm sewer samples at relatively low levels as compared to groundwater. Based on the risk factor calculations, PCP and 2,3,7,8-TCDD (Equivalent) appear to be the primary chemicals of potential concern in storm sewer surface water. Of the inorganic compounds detected in storm sewer surface water samples, arsenic and manganese appeared to be the primary chemicals of potential concern.

The TICs detected in surface water at the Havertown PCP site are presented in Table 6-7. The majority of the TICs consisted of alkyl benzenes and PAHs. The presence of these compounds is consistent with the disposal history of the site.

6.1.2.5 Sediments

Sediment samples were collected from the same six sites in Naylors Run as were the surface water samples. In addition, two sediment samples were collected from storm sewers. One storm sewer sediment sample was collected on the north east corner of the NWP property close to Naylors Run, while the other was collected behind the PCG plant, approximately 250 feet before it empties into Naylors Run. Many of the organic chemicals, particularly PAHs, were found at higher concentrations in Naylors Run sediment samples as compared to storm sewer samples. In addition, some of the highest detected concentrations of PAHs were found upstream from the catch basin. This may indicate that surface water run off from the NWP site may be a significant source of PAH contamination in Naylors Run.

The second round of sediment data was collected primarily to fill data gaps for conducting the aquatic ecological assessment. These data were included in the

Table 6-6

Summary of Chemicals Detected in
Storm Sewer Surface water at the Havertown PCP Site

| Compound | SF Risk Factor (a) | RfD Risk Factor (b) | Human Nutrient (c) | Concentration Data (Units: ug/L)(d) Frequency of Detection | Minimum Detected | Geometric Mean | Maximum Detected |
|---|---|---|---|---|---|---|---|
| Organics: | | | | | | | |
| Acetone | -- | <1% | No | 1/2 | 140.0 | NC | 140 |
| •Benzene | 1.0% | -- | No | 1/3 | 120.0 | 9.1 | 120.0 |
| Benzoic acid | -- | <1% | No | 2/3 | 9.0 | NC | 18.0 |
| Benzyl alcohol | -- | <1% | No | 1/3 | 8.0 | NC | 8.0 |
| Bromodichloromethane | <1% | <1% | No | 1/3 | 1.0 | NC | 1.0 |
| 2-Butanone | -- | <1% | No | 1/2 | 80.0 | NC | 80.0 |
| Chloroform | <1% | <1% | No | 1/3 | 1.0 | NC | 1.0 |
| Ethyl Benzene | -- | <1% | No | 1/3 | 110.0 | 8.8 | 110.0 |
| •2-Methylnaphthalene | -- | -- | No | 1/3 | 110.0 | 14.0 | 110.0 |
| Naphthalene | -- | <1% | No | 1/3 | 2.5 | NC | 2.5 |
| •Phenanthrene | -- | -- | No | 1/3 | 19.0 | 7.9 | 19.0 |
| •Pentachlorophenol | 68.9% | 1.0% | No | 1/3 | 2,100.0 | 110.0 | 2,100.0 |
| 2,3,7,8-TCDD (Equivalent) | 28.7% | 96.8% | No | 1/3 | 0.703ppt | NC | 0.203ppt |
| Trichloroethene | <1% | <1% | No | 1/3 | 16.0 | 4.6 | 16.0 |
| Xylenes (total) | -- | <1% | No | 2/3 | 2.5 | 15.0 | 500.0 |
| Inorganics: | | | | | | | |
| •Aluminum | -- | -- | No | 2/2 | 129.0 | NC | |
| •Arsenic | 1.4% | <1% | No | 1/4 | 3.0 | 1.3 | 3.0 |
| •Barium | -- | <1% | No | 4/4 | 30.0 | 55.0 | 113.0 |
| Calcium | -- | -- | Yes | 4/4 | 19,500.0 | 31,000.0 | 57,100.0 |
| Chromium | -- | <1% | No | 2/3 | 4.0 | 3.6 | 5.9 |
| •Cobalt | -- | -- | No | 1/3 | 60.2 | 3.9 | 60.2 |
| Copper | -- | <1% | Yes | 2/4 | 14.9 | 5.4 | 24.7 |
| Iron | -- | -- | Yes | 4/4 | 349.0 | 1,800.0 | 13,800.0 |
| •Lead | -- | -- | No | 2/4 | 3.2 | 2.1 | 5.9 |
| Magnesium | -- | -- | Yes | 4/4 | 9,330.0 | 11,000.0 | 12,600.0 |
| •Manganese | -- | 2.0% | No | 4/4 | 77.1 | 630.0 | 14,300.0 |
| Potassium | -- | -- | Yes | 4/4 | 1,220.0 | 2,800.0 | 5,160.0 |
| Silver | -- | <1% | No | 1/4 | 4.9 | 2.5 | 4.9 |
| Sodium | -- | -- | Yes | 4/4 | 20,900.0 | 26,000.0 | 51,300.0 |
| Zinc | -- | <1% | Yes | 2/2 | 103.0 | NC | 175.0 |

NC Not calculated
• Chemicals of potential concern
-- No toxicity criteria
(a) Percent contribution of carcinogenic risk based on the exposure point concentration and the slope factor (see text for further discussion).
(b) Percent contribution of non-carcinogenic risk based on the exposure point concentration and the RfD (see text for further discussion).
(c) Compound is an essential human nutrient. Concentrations of compound would result in exposures that are less than the Recommended Daily Allowance (RDA).
(d) Data analyzed according to data screening procedures outlined in Section 6.1.2.1. Frequency of detection is the number of detected concentrations divided by the number of samples (which may vary due to blank related contamination). Minimum and maximum concentration may be the average of duplicate samples.

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Table 6-7

Tentatively Identified Compounds (TICs)
Detected in Surface Water at the Havertown PCP Site

| TIC | Range of Concentrations (ug/L) |
|---|---|
| Alkenylbenzene | 10-18 |
| 9,10-Anthracenedione | 110-200 |
| 1-Cyclohexyl-2-propanone | 220 |
| 4H-Cyclopenta(def)phenanthrene | 260-500 |
| Dimethybenzene | 11 |
| Dimethylbenzenemethanol | 26 |
| bis(Dimethylethyl)phenol | 19-23 |
| Dimethylethylphenol isomer | 10-12 |
| N,N-Dimetnylmethanamine | 1-6 |
| Dimetnylnapnthalene | 16-31 |
| Dodecanoic acid | 58-210 |
| 2-Ethyl-1-hexanone | 16 |
| 1-(4-Ethylphenyl)-ethanone | 9.8-17 |
| Fatty acid | 4.2-69 |
| Hexadecanoic acid | 310-1100 |
| 1,4-Methanonapnthalen-9-ol | 12 |
| 2-Methylbenzenemethanol | 4.4-19 |
| Molecular Sulfur | 690 |
| Propylbenzene | 6.0-8.5 |
| Tetrachlorobenzenediol | 13-30 |
| Tetrachlorophenol | 18-48 |
| Tetradecanoic acid | 87 |
| Trimethylbenzene | 7.4-34 |
| Trimethylbenzonemethanol | 5.7-43 |
| Trimethylnaphthalene | 5.8-18 |
| Unknown | 4.8-420 |
| Unknown alcohol | 24-31 |
| Unknown Aldol | 150-610 |
| Unknown Chlorinated Organic | 22-32 |
| Unknown hydrocarbon | 4.1-250 |
| Unknown PAH | 150-610 |

ecological assessment. In general, significantly lower concentrations of benzo(a)pyrene (Equivalent), dioxin, and heavy metals were found further downstream.

Naylors Run Sediment - Table 6-8 presents chemicals detected in sediment from Naylors Run. Chemicals of potential concern identified in Table 6-8 were selected based on the criteria presented in Section 6.1.2.2. Based on the carcinogenic risk factor, benzo(a)pyrene (Equivalent), 2,3,7,8-TCDD (Equivalent), and arsenic appeared to be the primary chemicals of potential concern in sediment samples. The highest detected concentrations of benzo(a)pyrene (Equivalent) was detected in Naylors Run directly downstream from the Eagle Road over-pass. The highest detected concentrations of 2,3,7,8-TCDD (Equivalents) were detected along Naylors Run in the catch basin area. Based on the noncarcinogenic risk factor, 2,3,7,8-TCDD (Equivalent) and several inorganic chemicals appeared to be the primary chemicals of concern. The maximum detected concentrations of the inorganic chemicals of potential concern were found at different locations along Naylors Run.

Storm Sewer Sediment - Table 6-9 presents chemicals detected in sediment from the storm sewers. Chemicals of potential concern identified in Table 6-8 were selected based on the criteria presented in Section 6.1.2.2. Based on the risk factors presented in Table 6-9, arsenic appears to be the primary chemical of potential concern in storm sewer sediments. Benzo(a)pyrene (Equivalent) and chromium also had significant risk factors. It should be noted, however, that storm sewer sediment samples were not analyzed for dioxin and furan isomers. Dioxin and furan are most likely present in storm sewer sediments given the levels detected in storm sewer surface water. Thus, 2,3,7,8-TCDD (Equivalent) actually may be the primary chemical of concern in storm sewer sediment. There is no complete exposure pathway associated with direct contact with storm sewer sediments. Although storm sewer sediments may act as a potential source to Naylors Run, sediments in Naylors Run were considered the point of exposure for evaluating risk from sediments (dioxin was analyzed in Naylors Run sediment).

Table 6-8

Summary of Chemicals Detected in
Naylors Run Sediment at the Havertown PCP Site

| Compound | SF Risk Factor (a) | RfD Risk Factor (b) | Human Nutrient (c) | Concentration Data (d) Organics: ug/Kg, Inorganics: mg/Kg Frequency of Detection | Minimum Detected | Geometric Mean | Maximum Detecte |
|---|---|---|---|---|---|---|---|
| Organics: | | | | | | | |
| Acetone | -- | <1% | No | 2/4 | 20.0 | 26.0 | 650. |
| Aldrin | <1% | <1% | No | 1/6 | 22.5 | 13.0 | 22. |
| beta-BHC | <1% | -- | No | 2/6 | 18.5 | 16.0 | 35. |
| 2-Butanone | -- | <1% | No | 2/6 | 17.0 | 12.0 | 120. |
| *alpha-Chlordane | -- | -- | No | 1/6 | 110.0 | 99.0 | 110. |
| *gamma-Chlordane | -- | -- | No | 1/6 | 130.0 | 100.0 | 130. |
| *Chlordane (total) | <1% | 1% | No | 1/6 | 240.0 | 200.0 | 240. |
| bis(2-Chloroethyl)ether | <1% | -- | No | 5/6 | 910.0 | 1,000.0 | 3,200. |
| 4,4'-DDD | <1% | <1% | No | 2/6 | 43.0 | 28.0 | 51. |
| *Dibenzofuran | -- | -- | No | 1/6 | 900.0 | 460.0 | 900. |
| 1,2-Dichlorobenzene | -- | <1% | No | 5/6 | 2,300.0 | 3,500.0 | 55,000. |
| 1,4-Dichlorobenzene | <1% | <1% | No | 1/6 | 1,100.0 | 480.0 | 1,100. |
| Dieldrin | <1% | <1% | No | 2/6 | 45.5 | 34.0 | 75. |
| *Endosulfan Sulfate | -- | -- | No | 1/6 | 48.0 | 23.0 | 48. |
| Endrin | -- | <1% | No | 1/6 | 43.0 | 22.0 | 43. |
| Ethyl Benzene | -- | <1% | No | 1/6 | 5.0 | 3.3 | 5. |
| bis(2-Ethylhexyl)phthalate | <1% | <1% | No | 6/6 | 310.0 | 800.0 | 3,600. |
| Heptachlor | <1% | <1% | No | 1/6 | 160.0 | 19.0 | 160. |
| Methylene Chloride | <1% | <1% | No | 1/1 | 81.5 | NC | 81. |
| Polycyclic aromatic Hydrocarbons | | | | | | | |
| *Acenaphthene | -- | -- | No | 1/6 | 1,300.0 | 570.0 | 1,300. |
| *Anthracene | -- | -- | No | 6/6 | 120.0 | 520.0 | 2,300. |
| *Benzo(a)anthracene | -- | -- | No | 6/6 | 290.0 | 1,700.0 | 7,500. |
| *Benzo(a)pyrene | -- | -- | No | 6/6 | 340.0 | 1,700.0 | 7,000. |
| *Benzo(a)pyrene (Equivalent)(f) | 82.1% | -- | No | 6/6 | 1,559.6 | 6,800.0 | 28,061. |
| *Benzo(b)fluoranthene | -- | -- | No | 6/6 | 380.0 | 2,200.0 | 11,000. |
| *Benzo(g,h,i)perylene | -- | -- | No | 3/6 | 487.5 | 550.0 | 1,100. |
| *Benzo(k)fluoranthene | -- | -- | No | 6/6 | 320.0 | 2,300.0 | 10,000. |
| *Chrysene | -- | -- | No | 6/6 | 440.0 | 2,500.0 | 11,000. |
| *Dibenzo(a,h)anthracene | -- | -- | No | 3/6 | 412.5 | 620.0 | 1,400. |
| *Fluoranthene | -- | 1.3% | No | 6/6 | 780.0 | 4,600.0 | 21,000. |
| Fluorene | -- | <1% | No | 3/6 | 300.0 | 570.0 | 1,800. |
| *Indeno(1,2,3-c,d)pyrene | -- | -- | No | 3/6 | 622.5 | 780.0 | 1,800. |
| *Phenanthrene | -- | -- | No | 6/6 | 570.0 | 3,500.0 | 20,000. |
| *Pyrene | -- | -- | No | 6/6 | 980.0 | 4,000.0 | 14,000. |
| *Pentachlorophenol | <1% | <1% | No | 4/6 | 810.0 | 1,400.0 | 3,000. |
| *2,3,7,8-TCDD (Equivalent)(g) | 4.7% | 29.5% | No | 6/6 | 0.003 | 0.053 | 0.11 |
| Toluene | -- | <1% | No | 1/6 | 8.0 | 3.7 | 8. |
| Xylenes (total) | -- | <1% | No | 3/6 | 4.0 | 7.9 | 88. |
| Inorganics: | | | | | | | |
| *Aluminum | -- | -- | No | 6/6 | 3,800.0 | 5,300.0 | 7,130. |
| *Antimony | -- | 8.8% | No | 5/6 | 5.5 | 9.4 | 14. |
| *Arsenic | 15.6% | 9.4% | No | 3/3 | 9.6 | 20.0 | 37. |
| *Barium | -- | 2.0% | No | 6/6 | 39.0 | 92.0 | 415. |
| Calcium | -- | -- | Yes | 6/6 | 13,600.0 | 26,000.0 | 65,400. |
| *Chromium | -- | 26.6% | No | 6/6 | 31.4 | 120.0 | 532. |
| *Cobalt | -- | -- | No | 6/6 | 7.1 | 13.0 | 29. |
| Copper | -- | <1% | Yes | 5/5 | 12.3 | 35.0 | 139. |
| Iron | -- | -- | Yes | 6/6 | 10,300.0 | 22,000.0 | 58,500. |
| *Lead | -- | -- | No | 5/6 | 12.0 | 49.0 | 694. |
| Magnesium | -- | -- | Yes | 6/6 | 6,310.0 | 15,000.0 | 34,100. |
| *Manganese | -- | 11.9% | No | 6/6 | 399.0 | 2,000.0 | 4,750. |
| Nickel | -- | <1% | No | 5/6 | 14.4 | 18.0 | 43. |
| Potassium | -- | -- | Yes | 6/6 | 1,055.0 | 1,600.0 | 2,160. |
| Sodium | -- | -- | Yes | 6/6 | 51.2 | 120.0 | 600. |
| *Thallium | -- | 3.6% | No | 2/6 | .8 | .5 | 1. |
| *Vanadium | -- | 4.2% | No | 6/6 | 20.5 | 50.0 | 118. |
| Zinc | -- | <1% | Yes | 6/6 | 95.5 | 140.0 | 243. |

NC Not calculated
* Chemical of potential concern
-- No toxicity criteria
(a) Percent contribution of carcinogenic risk based on the exposure point concentration and the slope factor (see text for further discussion).
(b) Percent contribution of non-carcinogenic risk based on the exposure point concentration and the RfD (see text for furth discussion).
(c) Compound is an essential human nutrient. Concentrations of compound would result in exposures that are less than the Recommended Daily Allowance (RDA).
(d) Data analyzed according to data screening procedures outlined in Section 6.1.2.1. Frequency of detection is the number detected concentrations divided by the number of samples (which may vary due to blank related contamination). Minimum a maximum concentration may be the average of duplicate samples.
(e) Concentrations for alpha-and gamma-chlordanes were summed to calculate total chlordanes.
(f) Concentrations of anthracene benzo(a)anthracene, benzo(a)phrene, benzo(b)flouranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene, and phyrene were summed using Toxicity Equivalency Factors (TEFs) to calculate total benzo(a)pyrene equivalents.
(g) 2,3,7,8-TCDD (equivalents) calculated by summing dioxin and furan congener data using 2,3,7,8-TCDD TEFS.

Table 6-9

Summary of Chemicals Detected in
Storm Sewer Sediment at the Havertown PCP Site

| Compound | SF Risk Factor (a) | RfD Risk Factor (b) | Human Nutrient (c) | Concentration Data (d) (Organics: ug/Kg, Inorganics: mg/K) Frequency of Detection | Minimum Detected | Maximum Detect |
|---|---|---|---|---|---|---|
| Organics: | | | | | | |
| Aldrin | <1% | <1% | No | 1/2 | 13.0 | 13 |
| Benzoic acid | -- | <1% | No | 1/2 | 270.0 | 270 |
| Butylbenzylphthalate | -- | <1% | No | 1/2 | 860.0 | 860.0 |
| alpha-Chlordane | -- | -- | No | 1/2 | 22.0 | 22.0 |
| gamma-Chlordane | -- | -- | No | 1/2 | 29.0 | 29 |
| Chlordane (total)(e) | <1% | <1% | No | 1/2 | 51.0 | 51 |
| •Dibenzofuran | -- | -- | No | 1/2 | 69.0 | 69.0 |
| bis(2-Ethylhexyl)phthalate | <1% | <1% | No | 2/2 | 260.0 | 720.0 |
| Polycyclic aromatic hydrocarbons | | | | | | |
| •Acenaphthene | -- | -- | No | 1/2 | 95.0 | 95 |
| •Acenaphthylene | -- | -- | No | 1/2 | 280.0 | 280.0 |
| •Anthracene | -- | -- | No | 2/2 | 270.0 | 470.0 |
| •Benzo(a)anthracene | -- | -- | No | 2/2 | 710.0 | 1,200 |
| •Benzo(a)pyrene | -- | -- | No | 2/2 | 620.0 | 840 |
| •Benzo(a)pyrene (Equivalent) | 5.5% | -- | No | 2/2 | 2,011.5 | 3,671. |
| •Benzo(b)fluorantnene | -- | -- | No | 2/2 | 620.0 | 1,300.0 |
| •Benzo(g,h,i)perylene | -- | -- | No | 2/2 | 370.0 | 970 |
| •Benzo(k)fluoranthene | -- | -- | No | 2/2 | 520.0 | 1,400 |
| •Chrysene | -- | -- | No | 2/2 | 890.0 | 1,600 |
| •Dibenzo(a,h)anthracene | -- | -- | No | 2/2 | 87.0 | 210.0 |
| Fluoranthene | -- | <1% | No | 2/2 | 1,200.0 | 1,900.0 |
| Fluorene | -- | <1% | No | 1/2 | 150.0 | 150 |
| •Indeno(1,2,3-c,d)pyrene | -- | -- | No | 2/2 | 440.0 | 1,100 |
| •Phenanthrene | -- | -- | No | 2/2 | 1,100.0 | 1,500.0 |
| •Pyrene | -- | -- | No | 2/2 | 960.0 | 1,[redacted]0 |
| •Pentachlorophenol | <1% | <1% | No | 1/2 | 20,000.0 | 20[redacted] |
| Trichloroethene | <1% | <1% | No | 2/2 | 3.0 | [redacted] |
| Inorganics: | | | | | | |
| •Aluminum | -- | -- | No | 2/2 | 7,320.0 | 12,900 |
| •Arsenic | 93.7% | 71.3% | No | 2/2 | 1.3 | 425 |
| Barium | -- | <1% | No | 2/2 | 115.0 | 137 |
| Beryllium | <1% | <1% | No | 2/2 | .6 | .8 |
| Cadmium | -- | <1% | No | 1/2 | 1.2 | 1.2 |
| Calcium | -- | -- | Yes | 2/2 | 61,900.0 | 71,600 |
| •Chromium | -- | 22.0% | No | 2/2 | 99.6 | 656 |
| •Cobalt | -- | -- | No | 2/2 | 4.7 | 10.7 |
| Copper | -- | <1% | Yes | 2/2 | 41.0 | 218.0 |
| Iron | -- | -- | Yes | 2/2 | 18,700.0 | 21,000 |
| •Lead | -- | -- | No | 2/2 | 30.3 | 226 |
| Magnesium | -- | -- | Yes | 2/2 | 27,700.0 | 44,900.0 |
| •Manganese | -- | 2.1% | No | 2/2 | 373.0 | 1,230.0 |
| •Mercury | -- | -- | No | 1/2 | .2 | |
| Nickel | -- | <1% | No | 2/2 | 8.9 | 23 |
| Potassium | -- | -- | Yes | 2/2 | 883.0 | 3,490.0 |
| Silver | -- | <1% | No | 2/2 | 1.9 | 2.5 |
| •Vanadium | -- | 1.1% | No | 2/2 | 35.8 | 47 |
| •Zinc | -- | 2.0% | Yes | 2/2 | 102.0 | 2,380 |

• Chemicals of potential concern

-- No toxicity criteria

(a) Percent contribution of carcinogenic risk based on the exposure point concentration and the slope factor (see text for further discussion).

(b) Percent contribution of non-carcinogenic risk based on the exposure point concentration and the RfD (see text for further discussion).

(c) Compound is an essential human nutrient. Concentrations of compound would result in exposures that are less than the Recommended Daily Allowance (RDA).

(d) Data analyzed according to data screening procedures outlined in Section 6.1.2.1. Frequency of detection is the nu[redacted] of detected concentrations divided by the number of samples (which may vary due to blank related contamination). Mini[redacted] and maximum concentration may be the average of duplicate samples.

(e) Concentrations of alpha-and gamma-chlordanes were summed to calculate total chlordanes.

(f) Concentrations of anthracene benzo(a)anthracene, benzo(a)phrene, benzo(b)flouranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene, and phyrene were summed using Toxicity Equivalency Factors (TEFs) to calculate total benzo(a)pyrene equivalents.

Therefore, the lack of dioxin data for storm sewer sediments does not impact the results or conclusions of the risk assessment.

The TICs detected in sediment at the Havertown PCP site are presented in Table 6-10. The majority of the TICs consisted of PAHs and chlorinated phenols. The chlorinated phenols and related compounds (i.e., alkyl phenols) may be associated with the breakdown of PCP.

6.1.2.6 Summary of Chemicals of Potential Concern

Table 6-11 lists the selected chemicals of potential concern for all media at the Havertown PCP site. Over forty chemicals were selected as chemicals of potential concern for the Havertown PCP site including volatile organic compounds, PCP, PAHs, pesticides, dioxins and furans, and inorganics. Of these chemicals, PCP, PAHs, and dioxins and furans appear to be the primary chemicals of potential concern in all media at the Havertown PCP site. Other chemicals selected as chemicals of potential concern in all media included: aluminum, arsenic, cobalt, and manganese. Several volatile organic compounds selected as chemicals of potential concern were detected only in groundwater including: 1,2-dichloroethene, trichloroethene, and vinyl chloride. The exclusive presence of these chemicals in groundwater may be due to their high water solubility, low affinity for binding to sediment particles, and potential volatilization from surface water to the air. The pesticides dieldrin and heptachlor epoxide were only detected in Naylors Run surface water. The majority of the PAHs were found only in sediment samples, probably due to their low water solubility and high affinity for binding to sediment particles. Several inorganic chemicals of potential concern including antimony, nickel, thallium, vanadium, and zinc were selected only in Naylors Run. It is uncertain whether these chemicals are actually associated with site related disposal. TICs identified in groundwater and surface water consisted of alkyl benzenes and PAHs. TIC PAHs and possible breakdown products of PCP were found in sediments. The presence of these TICs is consistent with the disposal history of the site.

Table 6-10

Tentatively Identified Compounds (TICs)
Detected in Sediment at the Havertown PCP Site

| TIC | Range of Concentrations (ug/kg) |
|---|---|
| Alkylnaphthalene | 2,000 |
| Benzo(b)naphtho-thiophene | 690 |
| Dibenzothiophene | 150 |
| Dimethylbiphenyl | 1,300 |
| Dimethylnaphthalene | 520-2,100 |
| Dimethylphenol | 290 |
| bis(1,1-dimethyl)phenol | 21 |
| Dimethyl PNA | 550-930 |
| Ethylmethylbenzene | 86 |
| Fatty acid | 180-1,300 |
| Ketone | 27-1,200 |
| Methylpropylbenzene | 80 |
| Methyl PNA | 480-550 |
| Sulfur (mol.)(S8) | 220-510 |
| Tetrachlorophenol | 1000-1,300 |
| 2,2,3,3,-Tetramethylbutane | 49-140 |
| Trichlorophenol | 680 |
| Trimethylbenzene | 6.7-34 |
| Trimethylnaphthalene | 850-1,500 |
| Unknown | 17-1,000 |
| Unknown alkylbenzene | 15-240 |
| Unknown hydrocarbon | 14-7,800 |
| Unknown ketone | 300-1,200 |
| Unknown PNA | 360-3,800 |
| Unknown Sterol | 3,900-4,000 |

Table 6-11
Summary of Chemicals of Potential Concern for the Havertown PCP Site

| | Ground-Water | Naylors Run | | Storm Sewer | |
|---|---|---|---|---|---|
| | | Surface Water | Sedi-ment | Surface Water | Sedi-ment |
| Organics: | | | | | |
| acenaphthene | X | | X | | X |
| acenaphthylene | X | | | | X |
| anthracene | X | | X | | X |
| benzene | X | | | X | |
| benzo(a)anthracene | X | | X | | X |
| benzo(a)pyrene | | | X | | X |
| benzo(a)pyrene (Equivalent) | X | X | X | | X |
| benzo(b)fluoranthene | | | X | | X |
| benzo(g,h,i)perylene | | | X | | X |
| benzo(k)fluoranthene | | | X | | X |
| bis(2-ethylhexyl)phthalate | X | | | | |
| chlordane(Total) | | | X | | |
| chrysene | X | | X | | X |
| dibenzo(a,h)anthracene | | | X | | X |
| dibenzofuran | X | | X | | X |
| 1,2-dichloroethene | X | | | | |
| dieldrin | | X | | | |
| endosulfan sulfate | | | X | | |
| fluoranthene | X | | X | | |
| heptachlor epoxide | | X | | | |
| indeno(1,2,3-c,d)Pyrene | | | X | | X |
| naphthalene | X | | | | |
| 2-methylnaphthalene | X | | | X | |
| pentachlorophenol | X | X | X | X | X |
| phenanthrene | X | | X | X | X |
| pyrene | X | X | X | | X |
| 2,3,7,8-TCDD (Equivalent) | X | X | X | X | |
| trichloroethene | X | | | | |
| vinyl chloride | X | | | | |

Table 6-11(Cont.)
Summary of Chemicals of Potential Concern for the Havertown PCP Site

| | Ground -Water | Naylors Run | | Storm Sewer | |
|---|---|---|---|---|---|
| | | Surface Water | Sedi- ment | Surface Water | Sedi- ment |
| Inorganics: | | | | | |
| aluminum | X | X | X | X | X |
| antimony | | | X | | |
| arsenic | X | | X | X | X |
| barium | | | X | X | |
| chromium | | | X | | X |
| cobalt | X | X | X | X | X |
| lead | | X | X | X | X |
| manganese | X | X | X | X | X |
| mercury | | | | | X |
| nickel | | | X | | |
| thallium | | X | X | | |
| vanadium | | | X | | X |
| zinc | | | | | X |

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### 6.1.3 Exposure Assessment

This section quantifies the magnitude, frequency, and duration of exposure from chemicals released to groundwater, surface water, and sediment from the Havertown PCP site. The exposure assessment for the Havertown PCP site was conducted in accordance with available EPA (1990a, 1989a,b,c, and 1988a) guidance.

The first step in the exposure assessment process is characterizing the environmental setting of the site. The environmental setting consists of the physical environment and potentially exposed populations. The physical environment for the Havertown PCP site was discussed in Section 2 of this RI report. The environmental setting of the Havertown PCP site will be further discussed in Section 6.1.3.1 of the baseline risk assessment.

Identifying exposure pathways is the second step of the exposure assessment process which includes: 1) evaluating chemical sources, release mechanisms, and transport; 2) identifying possible exposure points; and 3) identifying the exposure routes. Chemical sources, release mechanisms, and transport were discussed in Section 5 of this report. Section 6.1.3.1, of this RI report, reviews possible exposure routes and identifies the exposure pathways of concern.

The final step in the exposure assessment process is quantifying exposure for the identified exposure routes for the reasonable maximum exposure (RME) case, as specified in the NCP (EPA 1990a). Exposure is quantified in Sections 6.1.3.2 and 6.1.3.3 of this report for the exposure pathways of concern. Section 6.1.3.2 describes the methods used to estimate exposure point concentrations and quantifies exposure point concentrations for chemicals of potential concern identified in Section 6.1.2. Section 6.1.3.3 describes the methods used to estimate exposure (i.e., chronic daily intakes [CDIs]) for the exposure pathways evaluated in this report. The CDIs will be used in conjunction with toxicity criteria (identified in Section 6.1.4) to characterize the potential risk

associated with the Havertown PCP site under current and future land-use conditions.

Media evaluated in the exposure assessment include: groundwater, surface water, sediments, and air. Exposure pathways associated with contaminated soil and releases from soil were not within the scope of the Havertown PCP baseline risk assessment. These pathways were evaluated in the Phase I RI baseline risk assessment prepared by Greeley-Polhemus Group, Inc. (1989). Exposure pathways evaluated in this report included direct contact with soils and inhalation of volatile organic compounds (VOCs) and dust released from soil. These pathways will not be reevaluated in this report.

6.1.3.1 Exposure Pathway Assessment

This section identifies "complete" exposure pathways which will be quantitatively evaluated in the Havertown PCP baseline risk assessment. A potentially "complete" exposure pathway has the following four characteristics:

1) mechanism of release (e.g., release of chemicals of potential concern from subsurface soil to groundwater);
2) transport media (e.g., transport of chemicals of potential concern in groundwater along a gradient);
3) point of exposure (e.g., chemicals of potential concern present in residential well); and
4) route of exposure (e.g., resident ingests groundwater from their private well).

Only "complete" exposure pathways which are both quantifiable and potentially significant are quantitatively evaluated in the baseline risk assessment. A summary of the "complete" exposure pathways evaluated under current and future land-use conditions of the Havertown PCP site are summarized in Table 6-12 and

Table 6-12

Potential Human Exposure Pathways for the Havertown PCP Site
Under Current Land-Use Conditions

| Exposure Media (a) | Exposure Point | Potential Receptor | Primary Exposure Routes | Exposure Pathway Complete? | Pathway Selected for Quantitative Evaluation? |
|---|---|---|---|---|---|
| Groundwater | No exposure point | | | No. There are no residential or industrial wells currently in use within the Havertown PCP study area. | No, pathway not complete. |
| Surface Water/ Sediments | Storm Sewer | Children playing; Workers Cleaning sumps | Dermal absorption and incidental ingestion of sediments and dermal absorption of chemicals in surface water | Yes. It is highly unlikely, however, that children would be exposed to storm sewer sediments to any significant extent. Worker exposure to storm sewer sediments would be very infrequent and contact minimized by protective clothing | No, due to low probability of significant exposure. |
| | Naylors Run | Children Playing | Dermal absorption and incidental ingestion of sediments and dermal absorption of chemicals in surface water | Yes, children may play in Naylors Run in the vicinity of the site | Yes |
| Air | On-site and in residential areas | Residents and workers | Inhalation of VOCs from groundwater seeps and storm sewer discharges (releases from soils evaluated in Phase I RI)(a) | Yes. It is unlikely, however, that significant releases of volatile would occur form surface water, given the minimal concentrations of VOCs in surface water. Dust would not be generated from contaminated sediments. | No, due to low probability of significant exposure. |
| Biota | Fish caught from Cobbs Creek | Recreational fisherman | Ingestion of contaminated fish tissue by fishermen and subsequent exposure to nursing infants via ingestion of breast milk from mothers that consume fish. | Yes. Chemicals of concern that may bioaccumulate to significant levels in fish tissue have been found. Fish tissue data from the NBS (EPA 1990b) were used in this assessment. Nursing infants also may be at risk if the mother consumes significant quantities of fish from Cobbs Creek. | Yes |

(a) Soil related exposure routes were evaluated in the Phase I RI baseline risk assessment (Greeley-Polhemus Group, 1989).

Table 6-13

Potential Human Exposure Pathways for the Havertown PCP Site
Under Future Land-Use Conditions

| Exposure Media (a) | Exposure Point | Potential Receptor | Primary Exposure Routes | Exposure Pathway Complete? | Pathway Selected for Quantitative Evaluation? |
|---|---|---|---|---|---|
| Groundwater | Hypothetical Residential Well | Resident | Ingestion of groundwater and inhalation of VOCs while showering. Also, nursing infants may be exposed to significant levels of dioxin from mothers that ingest groundwater. | Yes. If a well were installed in the primary areas of concern at the site, then significant exposure to chemicals of concern may occur via direct use of groundwater and indirect exposure to nursing infants that ingest breast milk from exposed mothers. Although the probability of this pathway occurring is low, it is evaluated primarily to justify potential remediation of groundwater for the site. | Yes |
| Surface Water/ Sediments | same as current land-use of the Havertown PCP site | | | | |
| Air | same as current land-use of the Havertown PCP site | | | | |
| Biota | same as current land-use of the Havertown PCP site | | | | |

(a) Soil related exposure routes were evaluated in the Phase I RI baseline risk assessment (Greeley-Polhemus Group, 1989).

Table 6-13, respectively. The environmental setting and pathway selection are discussed below.

Environmental Setting - The Havertown PCP site is located in Havertown, Haverford Township, Delaware county approximately 10 miles west of Philadelphia. The site is located in a mixture of commercial, industrial, and residential property. The 12 to 15 acre study area consists of the National Wood Preservers (NWP) facility, Philadelphia Chewing Gun Company (PCG) facility, and adjacent residential properties. Several playgrounds and schools are located within a one-mile radius of the Havertown PCP site.

Surface water run-off from the site eventually drains into Naylors Run which is located along the northern boundary of the site. Naylors Run also receives storm water flow from NWP drainage channels and storm water collection systems of PCG and Rittenhouse Circle. Naylors Run flows into Cobbs Creek approximately 4 miles southeast of the site near East Lansdowne. Cobbs Creek joins Darby Creek then flows through the Tinicum Wildlife Preserve prior to discharging into the Delaware River.

Groundwater flows in an easterly direction in the bedrock and overburden aquifers. Depth to groundwater may range from 0.5 feet in the vicinity of the Rittenhouse Circle to 23 feet at the Youngs Produce Store. Groundwater may provide base flow of Naylors Run. In addition, groundwater may discharge to cracks in the storm sewer system which discharge directly to Naylors Run.

Groundwater is currently not used as a source of drinking water in the vicinity of the Havertown PCP site. No active residential, municipal or industrial wells are known to be installed within a mile of the site based on available records from Havertown Township, Delaware County, State or Federal agencies. Residents in the area receive their water from the City of Havertown.

Approximately, 18,000 individuals live within one mile of the Havertown PCP site. Within a quarter mile of the site are approximately 350 residential properties. About a dozen workers and 12 residential properties are located within 500 feet of the site.

## Exposure Pathways Under Current Land-Use Conditions

Groundwater - As previously discussed, no residential, municipal, or industrial wells are located within 1 mile of the Havertown PCP site. Residents in the immediate vicinity of the site and presumably residents located farther downgradient use municipal water supplied by the City of Havertown. Therefore, there is no "complete" exposure pathway associated with direct contact with groundwater at the Havertown PCP site. However, groundwater may be used in the future as a potential drinking water resource (although unlikely) and will be evaluated as a hypothetical scenario in this report (as discussed further in the sections to follow).

Surface Water/Sediments - There are several residential properties immediately adjacent to the Havertown PCP site. In addition, several playgrounds and schools are located within 1 mile of the site. Therefore, it is likely that children may come in direct contact with sediments and surface water at Naylors Run. The catch basin, which had some of the highest detected concentrations of certain chemicals of potential concern, is currently fenced and locked. This may prevent access to potentially more contaminated storm sewer discharge. However, it should be noted that relatively high concentrations of the chemicals of concern that significantly contributed to overall risk (i.e., benzo(a)pyrene [Equivalent], PCP, and 2,3,7,8-TCDD [Equivalent]) were detected upstream and downstream of the fenced catch basin. Therefore, children that play in Naylors Run may be exposed to chemicals of potential concern via incidental ingestion and dermal contact with sediments and dermal contact with surface water. Exposure from incidental ingestion of surface water is considered negligible during

playing activities. Workers at in the vicinity of the Havertown PCP are not expected to come in direct contact with Naylors Run surface water or sediments to any significant degree.

In general, storm sewer water and sediments had higher detected concentrations of chemicals of potential concern than surface water and sediments from Naylors Run. It is highly unlikely, however, that children or workers would be exposed to storm sewer water or sediments. Therefore, this pathway was not quantitatively evaluated in this report.

Air - VOCs detected in surface water may be released to the air. VOCs detected in surface water included acetone, benzene, 1,2-dichloroethene, toluene, and trichloroethene. These chemicals were detected only at the catch basin at concentrations often below the contract required quantification limit (CRQL). After release to the air, VOCs would be significantly diluted at potential downwind exposure points (i.e., nearby residents). It is unlikely that residents would be exposed to significant levels of VOCs released from surface water.

In the immediate vicinity of the catch basin, however, potential exposure and impacts associated with emissions from storm sewer discharges cannot be ruled out. In 1981, workers conducting field investigations at Naylors Run in the immediate vicinity of the storm sewer discharge suffered irritations to the eyes, skin, and mucous membranes from apparent volatilization of chemicals from the discharge. Discharge of highly contaminated storm sewer water to Naylors Run, however, has been minimized by the catch basin and other remedial activities. In addition, concentrations of chemicals of potential concern in storm water have presumably decreased since the 1981 incident given the levels of VOCs found in storm sewer discharge and the catch basin. Therefore, potential exposure to VOCs via inhalation does not appear to be a significant pathway of concern.

Biota - Several of the chemicals detected in surface water and sediments

including PAHs, PCP, dioxins, and furans may bioaccumulate in the food chain. A bioassessment conducted for the Havertown PCP site revealed that game fish may inhabit deep pools along Cobbs Creek. No viable populations of game fish, however, were found in Naylors Run (see Section 6.2 for further discussion). Recreational fisherman who catch fish from Cobbs Creek may be exposed to chemicals of potential concern via ingestion of fish tissue. In addition, nursing infants may be indirectly exposed to chemicals of potential concern in fish tissue if the mother ingests significant quantities of fish from Cobbs Creek over several years prior to nursing.

Exposure Pathways Under Future Land-Use Conditions

Exposure pathways related to surface water, sediments, air, and biota are not suspected to change in the future. The exposure pathways evaluated under current land-use conditions for these media should be representative and sufficiently protective of future land-use of the Havertown PCP site. Exposure pathways related to future use of groundwater at the Havertown PCP site are the only additional pathways evaluated in the baseline risk assessment. If groundwater at the site were used as a source of water in the future, then residents may be exposed to chemicals of potential concern via ingestion. In addition, use of groundwater for bathing, showering, and cooking would result in exposure via inhalation of VOCs and dermal absorption. In general, exposure via dermal contact is insignificant compared to exposure via ingestion and inhalation while showering; therefore, this exposure route will not be evaluated quantitatively in this risk assessment. Also, nursing infants may be indirectly exposed to chemicals of potential concern in groundwater via ingestion of breast-milk from mothers that use groundwater at the site as a source of water for drinking, showering, etc.

It should be emphasized that it is highly unlikely that residents would actually use groundwater in the vicinity of the Havertown PCP site as a source of drinking

water in the future. Residents in the area currently use municipal water provided by the City of Havertown, and residential homes constructed in the future would likely be hooked-up to the city water supply system. In addition, further commercial and industrial development would likely use water supplied by the City of Havertown. However, future use of groundwater was evaluated quantitatively in this report primarily to justify further restrictions on groundwater use and in order to provide the basis for making risk management decisions concerning remediation of groundwater at the Havertown PCP site.

Summary of Exposure Pathways to be Quantitatively Evaluated

The following current land-use exposure pathways will be quantitatively evaluated in this report:

- direct contact with surface water and sediments by children playing in Naylors Run;
- ingestion of fish caught from Cobbs Creek by recreational fisherman; and
- indirect exposure to nursing infants who ingest breast-milk from mothers which are exposed to dioxin via ingestion of fish from Cobbs Creek.

The following future land-use exposure pathways will be quantitatively evaluated in this report:

- ingestion of groundwater at the Havertown PCP site by future hypothetical residents;
- inhalation of VOCs while showering by future hypothetical residents that use groundwater at the Havertown PCP site; and

- indirect exposure to nursing infants who ingest breast-milk from mothers which are exposed to dioxin via ingestion of groundwater.

6.1.3.2 Estimation of Exposure Point Concentrations

Methodology for Estimating Exposure Point Concentrations

To calculate exposure and ultimately risk, chemical-specific concentrations that a receptor could contact over the duration of the exposure period (i.e., exposure point concentrations) must be estimated. The exposure point concentration is defined as the average concentration contacted over the duration of the exposure period. This section describes the methods used to estimate exposure point concentrations for the exposure pathways quantitatively evaluated in this report.

In general, EPA (1989a) guidance recommends calculating the 95th upper confidence limit (UCL) on the arithmetic mean as the exposure point concentration using available monitoring data provided by the RI. EPA (1989a) guidance recommends applying a 95th UCL on the arithmetic mean concentration because of the uncertainty associated with available monitoring data. Two alternative methods for calculating the 95th UCL on the arithmetic mean have been recommended by EPA in Risk Assessment Guidance for Superfund (Gilbert 1987, as cited in EPA 1989a). One of the methods assumes that the individual chemical constituent concentrations are normally distributed and calculates a 95th UCL on the arithmetic mean from the t-distribution (Gilbert 1987). The other method, based on Land (1971, 1975), is used for chemical constituent concentration data that are lognormally distributed (Gilbert 1987).

The equation for calculating the 95th UCL on the arithmetic mean as presented in Land (1971, 1975) and Gilbert (1987) is presented below:

$$UCL(lognormal)_{0.95} = e^{\left(Y_1 + \frac{S_1^2}{2} + \frac{H_{0.95} * S_1}{\sqrt{N-1}}\right)}$$

where:

$UCL(lognormal)_{0.95}$ = The 95th UCL on the arithmetic mean concentration assuming a lognormal distribution;
e = natural log base (2.718);
$Y_1$ = arithmetic mean of the natural log transformed data;
$S_1$ = standard deviation of the natural log transformed data;
$H_{0.95}$ = tabular value which depends on the degrees of freedom, alpha, and standard deviation; and
N = sample size.

The equation for calculating the 95th UCL on the arithmetic mean assuming a normal distribution (Gilbert 1987) is presented below:

$$UCL(normal)_{0.95} = Y_n + t_{0.95} \frac{S_n^2}{\sqrt{N-1}}$$

where:

$UCL(normal)_{0.95}$ = The 95th UCL on the arithmetic mean assuming a normal distribution;
$Y_n$ = arithmetic mean of the untransformed data;
$S_n$ = standard deviation of the untransformed data; and
$t_{0.95}$ = t-statistic for a one-tailed confidence limit test with an alpha = 0.05; and
N = sample size.

In general, most chemical distributions in nature tend to be lognormally distributed except for abundant metals such as aluminum and iron (Connor and Shacklette 1975, Dean 1981, Esmen and Hammad 1977, and Ott 1988). Therefore,

between the two methods recommended by EPA, the method developed by Land (1971, 1975) should be used in most cases to calculate the 95th UCL on the arithmetic mean. In certain cases, however, the equation developed by Land (1971, 1975) will yield concentrations below the 95th UCL on the arithmetic mean calculated using the normal distribution equation. Generally, this occurs for inorganics which tend to be normally distributed. Thus, the 95th UCL on the arithmetic mean calculated using the normal distribution equation was used as the exposure point concentration in these cases.

EPA (1989a) guidance recommends using the maximum detected concentration as the exposure point concentration if the 95th UCL on the arithmetic mean exceeds the maximum detected concentration. The maximum concentration is often lower than the 95th UCL on the arithmetic mean calculated using the Land (1971, 1975) method when the sample size is small (e.g., less than 10 samples) and/or the chemical concentration distribution is highly positively skewed.

Estimation of Exposure Point Concentrations for Current Land-Use Pathways

Children Playing in Naylors Run - It was assumed that children may contact different locations along Naylors run while playing, over the duration of the exposure period (assumed to be 10 years). Based on this assumption, all of the surface water and sediment monitoring data collected from Naylors Run were used to estimate exposure point concentrations and exposure to children playing in Naylors run. Exposure point concentrations estimated for surface water and sediment from Naylors Run are presented in Tables 6-14 and 6-15, respectively.

Ingestion of Fish - For the fish ingestion pathway it is necessary to estimate the concentration of chemicals of potential concern from the site which may be present in fish tissue. No fish tissue samples, however, were collected as part of the field investigation for the Havertown PCP site. Modeling fish tissue concentrations using available surface water and sediment samples collected at

Table 6-14

Exposure Point Concentrations for the Chemicals of Concern
Detected in Surface Water from Naylors Run
(Concentrations in ug/L)

| | Average Concentration | 95th UCL on the Arithmetic Mean | | Maximum Concentration | Exposure Point Concentratic |
|---|---|---|---|---|---|
| | | Normal | Log-Normal | | |
| Naylors Run | | | | | |
| Organics: | | | | | |
| Dieldrin | .1 | .2 | .6 | .3 | .3 (a) |
| Heptachlor Epoxide | .2 | .5 | 38.0 | .8 | .8 (a) |
| Benzo(a)pyrene (Equivalent) | .3 | NC | NC | .3 | .3 (a) |
| Pentachlorophenol | 430.0 | 890.0 | >1,000,000.0 | 1,200.0 | 1,200.0 (a) |
| 2,3,7,8-TCDD (Equivalent) | 6.0E-5 | NC | NC | 3.0E-4 | 3.0E-4 ( |
| Inorganics: | | | | | |
| Manganese | 5,300.0 | 9,600.0 | >1,000,000.0 | 10,100.0 | 10,100.0 (a) |
| Thallium | 2.1 | 3.1 | 5.7 | 3.3 | 3.3 (a) |

NC Not calculated
(a) The lognormal 95th UCL on the arithmetic mean concentration exceeded the maximum detected concentration, or there were n enough samples (i.e., <3) available for estimating the 95th UCL on the arithmetic mean. Therefore, the maxim concentration was used as the exposure point concentration.

Table 6-15

Exposure Point Concentrations for the Chemicals of Concern
Detected in Sediment from Naylors Run
(Organic Concentrations: ug/kg; Inorganic Concentrations: mg/kg)

| | Average Concentration | 95th UCL on the Arithmetic Mean | | Maximum Concentration | Exposure Point Concentration |
|---|---|---|---|---|---|
| | | Normal | Log-Normal | | |
| Organics: | | | | | |
| Chlordane (total) | 200.0 | 220.0 | 230.0 | 240.0 | 230.0 (a) |
| Benzo(a)pyrene (Equivalent) | 11,000.0 | 19,000.0 | 110,000.0 | 28,061.7 | 28,061.7 (b) |
| Fluoranthene | 7,700.0 | 14,000.0 | 150,000.0 | 21,000.0 | 21,000.0 (b) |
| Pentachlorophenol | 1,600.0 | 2,500.0 | 4,200.0 | 3,000.0 | 3,000.0 (b) |
| 2,3,7,8-TCDD (Equivalent) | 0.06 | NC | NC | 0.118 | 0.118 (b) |
| Inorganics: | | | | | |
| Antimony | 10.0 | 13.0 | 15.0 | 14.1 | 14.1 (b) |
| Arsenic | 23.0 | 47.0 | 2,100.0 | 37.6 | 37.6 (b) |
| Barium | 130.0 | 250.0 | 580.0 | 415.0 | 415.0 (b) |
| Chromium | 230.0 | 420.0 | 9,600.0 | 532.0 | 532.0 (b) |
| Manganese | 2,600.0 | 4,000.0 | 14,000.0 | 4,750.0 | 4,750.0 (b) |
| Nickel | 20.0 | 30.0 | 33.0 | 43.7 | 33.0 (a) |
| Thallium | .6 | .9 | 1.7 | 1.0 | 1.0 (b) |
| Vanadium | 59.0 | 88.0 | 160.0 | 118.0 | 118.0 (b) |

NC Not calculated
(a) Exposure point concentration based on the 95th UCL on the arithmetic mean concentration derived using Land (1971, 1975 which assumes that the distribution is lognormal.
(b) The lognormal 95th UCL on the arithmetic mean concentration exceeded the maximum detected concentration, or there were not enough samples (i.e., <3) available for estimating the 95th UCL on the arithmetic mean; therefore, the maximum concentration was used as the exposure point concentration.

the headwaters of Naylors Run may be too conservative, given that viable populations of game were not found in Naylors Run. The lack of game fish is probably due to the size of the stream; however, the presence of chemicals that may inhibit development of an aquatic food web cannot be ruled out. Viable fish populations, however, were found in pools located along Cobbs Creek.
As part of the National Bioaccumulation Survey (NBS) conducted by EPA (1990d), seven fish samples, including 2 black bullhead and 5 white sucker, were collected from Cobbs Creek approximately 5 miles downstream of the Havertown PCP site. The black bullhead samples were analyzed as fillets while the white sucker samples were analyzed as whole body. Possible sources of chemicals present in fish tissue sampled from Cobbs Creek include: Havertown PCP site, non-point sources (e.g., agricultural pesticide spraying), and a landfill. Samples of fish also were collected from Schuylkill River for which Cobbs Creek is a tributary. The samples from Schuylkill River, however, were further downstream of the site and several other sources may contribute to chemicals present in fish tissue; therefore, these samples were not included in the Havertown PCP baseline risk assessment.

The fish samples were analyzed for chemicals that tend to bioaccumulate in fish tissue including dioxins and furans, heavy metals, pesticides, and PCBs. Of the chemicals detected in fish tissue, only chemicals of potential concern detected in surface water and sediments in Naylors Run directly downstream of the site were included in this assessment. This selection process was performed in an attempt to delineate the contribution of the Havertown PCP site to the potential risk associated with ingesting fish from Cobbs Creek (however it is uncertain whether the site is the actual source). This is necessary in order that appropriate risk management decisions can be made with regard to remediation of Naylors Run and storm sewer discharges from the Havertown PCP site. Therefore, this assessment does not present total exposure and risks associated with

ingestion of fish. Exposure point concentrations for fish tissue samples are presented in Table 6-16. Chlordane, dieldrin, heptachlor epoxide, and dioxins were detected in both fish tissue samples taken from Cobbs Creek and in either the sediments or surface water from Naylors Run. It should be noted, however, that PCP and PAHs which are chemicals of potential concern at the Havertown PCP site and may bioaccumulate in the food chain, were not included in the NBS. Therefore, the potential exposure and risk associated with ingestion of fish may be underestimated.

Nursing Infants - A pharmacokinetic model was used to estimate indirect exposure to nursing infants from ingestion of contaminated breast-milk from mothers who are exposed via ingestion of fish tissue. Based on the results of the model, exposure to nursing infants is directly proportional to the exposure to the mother. Therefore, exposure point concentrations were not estimated for the nursing infant exposure pathway.

## Estimation of Exposure Point Concentrations for Future Land-Use Pathways

For the future land-use groundwater ingestion pathway, it was assumed that a hypothetical resident may install a well anywhere at the site. If a well were installed near a "hot spot" location, then an individual may contact relatively high concentrations in the general vicinity of the well and not an average concentration from the entire study area. Therefore, it would not be appropriate to use all of the groundwater data collected at the Havertown PCP site to estimate exposure point concentrations for the groundwater ingestion pathway (in contrast to estimating exposure to children who play in Naylors Run and may contact different locations over the duration of exposure). In order that potential exposure will not be underestimated, EPA Region III (1991a) recommends selecting three wells with groundwater contamination which are indicative of site contamination. EPA recommends using several sampling rounds from these wells in

Table 6-16

Chemicals of Potential Concern
Detected in Fish Tissue Sampled from
Cobbs Creek (a)

| Chemical (b) | Concentration in ug/kg | |
|---|---|---|
| | Black Bullhead (c) | White Sucker (d) |
| Chlordane (total) | 59.0 | 238 |
| Dieldrin | 63 | 450 |
| Heptachlor Epoxide | 8.6 | 37 |
| 2,3,7,8-TCDD (Equivalent) | 0.0013 | 0.007 |

(a) Fish samples collected from Cobbs Creek approximately 5 miles downstream of the Havertown PCP Site. Samples collected as part of the National Bioaccumulation Study (NBS) (EPA, 1990b).
(b) Only data for chemicals of potential concern which may be associated with the site were summarized. Note that PCP and PAHs were not analyzed as part of the NBS survey.
(c) Results represent composite of fillets from 2 black bullhead fish.
(d) Results represent composite of whole body samples from 5 white suckers.

order that the seasonal influence on contamination levels may be characterized. The 95th UCL on the arithmetic mean is estimated using the equations previously discussed using all of the data collected from the three representative wells.

The Havertown PCP sampling plan, however, was developed and implemented prior to release of the EPA Region III recommended approach for estimating exposure to groundwater. Only two sampling rounds were collected from the wells at the Havertown PCP site. The first round of sampling was used for screening purposes in order to identify areas that may need further groundwater contamination delineation. Groundwater samples were not collected at all well locations during the first round of sampling. In addition, the data was not validated (since it was used for screening purposes) and therefore could not be used in the risk assessment. Only the second round of sampling were available for quantitative use in the risk assessment. The first round of sampling, as well as historical data, were used qualitatively in the RI to evaluate potential fluctuations and/or trends in groundwater contamination.

For estimating exposure point concentrations for the Havertown PCP site, the 95th UCL on the arithmetic mean was calculated using available data from the three most contaminated well locations which include: HAV-2, HAV-4, and R-2. HAV-2, HAV-4, and R-2 are installed in the saprolite zone of the aquifer (HAV-2 and HAV-4 also are screened in the lower portion of the fill zone). These three well locations were selected because the highest detected concentrations of benzo(a)pyrene (Equivalent), naphthalene, PCP, and 2,3,7,8-TCDD (Equivalent) were found in these wells. These four chemicals contributed to more than 99 percent of the total carcinogenic and noncarcinogenic risk associated with groundwater use at the site. Certain chemicals of potential concern, however, were not detected in these three well locations including 1,2-dichloroethene, bis(2-ethyhexyl)phthalate, trichloroethene, vinyl chloride, and thallium. Data from all well locations installed in either the intermediate or deep portions of the aquifer were used to estimate exposure point concentrations for these chemicals.

Exposure point concentrations estimated for future-use of groundwater are presented in Table 6-17. As shown in this table, the 95th UCL on the arithmetic mean calculated using Land (1971, 1975) exceeded the maximum detected value for all chemicals that significantly contributed to risk (i.e., benzo(a)pyrene [Equivalent], naphthalene, PCP, and 2,3,7,8-TCDD [Equivalent]). Thus, the maximum detected value was used as the exposure point concentration for these chemicals. For comparison sake, exposure point concentrations were estimated using data from all monitoring wells at the site for these chemicals. Little difference was found between the estimated exposure point concentrations calculated using all the monitoring data versus data from the three well locations for the primary chemicals of concern in groundwater (i.e., benzo(a)pyrene [Equivalent], naphthalene, PCP, and 2,3,7,8-TCDD [Equivalent]). The exposure point concentrations presented in Table 6-17 for benzene, 1,2-dichloroethene (total), trichloroethene, and vinyl chloride also were used to estimate potential exposure via inhalation of VOCs while showering. As previously discussed, a pharmacokinetic model was used to estimate indirect exposure to nursing infants via ingestion of contaminated breast-milk from mothers who are exposed via ingestion of groundwater. Therefore, exposure pointconcentrations were not estimated for the nursing infant exposure pathway.

### 6.1.3.3 Estimation of Chronic Daily Intakes

This section describes the methods used to estimate exposure for the exposure pathways quantitatively evaluated under both current and future land-use conditions. According to the National Contingency Plan (NCP) (EPA 1990a), the exposure estimates should be based on a RME case. Exposure is referred to as the CDI which is expressed in terms of milligrams of contaminant contacted per kilogram of body weight per day (i.e., mg/kg/day). The CDI is calculated by combining exposure point concentrations and exposure parameter estimates using a chemical intake equation.

Table 6-17

Exposure Point Concentrations for the Chemicals of Concern
Detected in Groundwater for the Havertown PCP Site
(Concentrations in ug/L)

| | Average Concentration | 95th UCL on the Arithmetic Mean | | Maximum Concentration | Exposure Point Concentration |
|---|---|---|---|---|---|
| | | Normal | Log-Normal | | |
| Organics: | | | | | |
| Benzene | 180.0 | NC | NC | 230.0 | 230.0 (b) |
| 1,2-Dichloroethene (total) (d) | 58.0 | 92.0 | 280.0 | 245.0 | 245.0 (a) |
| bis(2-Ethylhexyl)phthalate (d) | 72.0 | 110.0 | 410.0 | 180.0 | 180.0 (b) |
| Benzo(a)pyrene (Equivalent) | 480.0 | NC | NC | 741.9 | 741.9 (b) |
| Fluoranthene | 810 | NC | NC | 810.0 | 810.0 (b) |
| Naphthalene | 12,000.0 | 32,000.0 | >1,000,000.0 | 24,000.0 | 24,000.0 (b) |
| Pentachlorophenol | 48,000.0 | 120,000.0 | >1,000,000.0 | 80,000.0 | 80,000.0 (b) |
| 2,3,7,8-TCDD (Equivalent) | 0.0009 | NC | NC | 0.17 | 0.17 (b) |
| Trichloroethene (d) | 79.0 | 130.0 | 490.0 | 465.0 | 465.0 |
| Vinyl Chloride (d) | 7.2 | 8.8 | 9.1 | 16.5 | 9.1 (a) |
| Inorganics: | | | | | |
| Arsenic | 11.0 | 30.0 | >1,000,000.0 | 22.7 | 22.7 (b) |
| Manganese | 20,000.0 | 25,000.0 | 26,000.0 | 22,600.0 | 22,600.0 (b) |
| Thallium (d) | 1.4 | 1.7 | 1.6 | 4.2 | 1.7 (c) |

NC Not calculated

(a) Exposure point concentration based on the 95th UCL on the arithmetic mean concentration derived using Land (1971, 1975, which assumes that the distribution is lognormal.

(b) The lognormal 95th UCL on the arithmetic mean concentration exceeded the maximum detected concentration, or there were not enough samples (i.e., <3) available for estimating the 95th UCL on the arithmetic mean. Therefore, the maximum concentration was used as the exposure point concentration.

(c) The chemical distribution was assumed to be normal; therefore, the normal 95th UCL on the arithmetic mean was used as the exposure point concentration.

(d) 1,2-Dichloroethene, bis(2-ethyhexyl)phthalate, vinyl chloride, and thallium were not detected in the most contaminated well locations (i.e., HAV-02, HAV-04, and R-2). Thus, data from all locations were used to estimate exposure point concentrations for these chemicals of potential concern.

The following sections describe the methodology used to estimate CDIs for the pathways quantitatively evaluated in this report. In addition, CDIs for chemicals of potential concern with available toxicity criteria are estimated for these exposure pathways.

Current Land-Use: Direct Contact with Surface Water by Children Playing in Naylors Run

Children may be exposed to chemicals of potential concern in surface water in Naylors Run while playing or wading. The estimated exposure to a chemical is based on the amount absorbed through the skin. The amount of surface water ingested is negligible during playing activities and; therefore, was not considered in this assessment.

Potential exposures to chemicals of potential concern in surface water via dermal absorption were calculated using the following equation:

$$CDI = (mg/kg/day) = \frac{(EPC)\,(SA)\,(PC)\,(ET)\,(EF)\,(ED)\,(CF_1)\,(CF_2)}{(BW)\,(AT)}$$

where:

CDI = Chronic Daily Intake (mg/kg/day);
EPC = Exposure Point Concentration (ug/L);
$CF_1$ = Conversion Factor ($10^{-3}$ mg/ug);
$CF_2$ = Conversion Factor (1 L/1000 $cm^3$);
SA = Skin Surface Area Available for Contact ($cm^2$);
PC = Dermal Permeability Constant (cm/hr);
ET = Exposure Time (hrs/day);
EF = Exposure Frequency (days/year);
ED = Exposure Duration (years);
BW = Body Weight (kg); and
AT = Averaging Time (days).

Exposure parameter values used to estimate exposure to children via contact with surface water are discussed below and summarized in Table 6-18.

EPC: The methods for estimating exposure point concentrations are presented in Section 6.1.3.2.

$CF_1$: This conversion factor adjusts the mass units.

$CF_2$: This conversion factor accounts for the volumetric unit conversion of 1 L to 100 $cm^3$.

SA: Approximately one-third of the total surface area of the hands, arms, and legs were assumed to directly contact surface water. Thus, approximately 1000 $cm^2$ of the body surface would contact contaminated surface water based on data presented in EPA (1985a,1989c) for children ages 2 to 12. The 50th percentile of the surface area of the hands, arms, and legs was used, rather than an upper-bound percentile, because it reflects the best estimate of the surface area for the individual with the 50th percentile body weight (EPA 1989a).

PC: The permeability constant reflects the movement of the chemical across the skin to the stratum corneum and into the bloodstream. Factors influencing dermal absorption from water include the nature of the compound, the presence of other agents which might facilitate the permeability of a chemical, as well as the properties of the skin itself (EPA 1988a). Chemical-specific permeability constant values are currently under review, as presented in the Superfund Exposure Assessment Manual (SEAM) (EPA 1988b), and are

Table 6-18

Exposure Parameters Used to Estimate
Exposure to Children via Direct Contact with Surface Water in Naylors Run

| Parameter | Value | Reference |
|---|---|---|
| $CF_1$ | $10^{-3}$ mg/ug | - - - |
| $CF_2$ | 1 L/1000 $cm^3$ | - - - |
| SA | 1000 $cm^2$ | (EPA, 1989a) |
| PC | 8.4 x $10^{-4}$ cm/hr | (Blank et al, 1984; EPA, 1989a) |
| ET | 2.6 hrs/day | (EPA, 1989a) |
| ED | 10 yrs | Assumed Value |
| EF | 125 days/yr | (EPA, 1989a) |
| AT | | |
| Carcinogens | 25,550 days | (EPA, 1989a) |
| Non-carcinogens | 3650 days | (EPA, 1989a) |
| BW | 25 kg | (EPA, 1985) |

not recommended for use in baseline risk assessments at this time (EPA 1989a). Currently, EPA (1989a) has recommended using the permeability of water of $8.4 \times 10^{-4}$ cm/hr for chemicals of potential concern (EPA 1989a, Blank et al. 1984). However, this method may underestimate skin permeability properties for some organic compounds (EPA 1989a), while overestimate the permeability of certain inorganic compounds.

ET: For the exposure time, it was assumed that contact with surface water during play activities would be similar to the national average of time spent swimming. The national average of time spent swimming is 2.6 hrs/day (EPA 1988a, 1989a).

EF: For the exposure frequency, it was assumed that children would play in Naylors Run three times per week for 10 weeks in the spring and fall when the temperature is above freezing (total of 60 days). In the summer months, accounting for warmer weather and schools being closed, children's exposure is considered to be up to five times per week for approximately thirteen weeks. Therefore, the exposure frequency would be 65 days during the summer. The total number of days exposed per year for the RME case was estimated to be 125 days/year (EPA 1989a).

ED: Children were assumed to play in Naylors Run between the ages of 2 and 12. Therefore, the exposure duration is 10 years. Children in this age group are more likely to engage in the activity outlined in this pathway than during other ages. In addition, children in this age group may have higher exposure (mg/kg/day) because of their lower body weights (kg) than older children which would have higher body weights.

BW: The mean body weight for both male and female children between the ages of 2 to 12 is approximately 25 kg (EPA 1985b).

AT: The averaging time is 10 years (exposure duration) x 365 days/year for noncarcinogens and 70 years (lifetime) x 365 days/year for carcinogens.

An example calculation of the CDI for carcinogens assuming an exposure point concentration of 1 ug/L is presented below:

$$CDI_{carcinogen} = \frac{(1\ ug/L)(10^{-3}\ mg/ug)(1000\ cm^3)(8.4 \times 10^{-4}\ cm/hr)(2.6\ hrs/day)(10\ yrs)(1\ L/1000\ cm^3)(125\ days/yr)}{(25550\ days)(25\ kg)}$$

$$CDI_{Carcinogens} = 4.3 \times 10^{-6}\ mg/kg/day$$

The CDI for noncarcinogens, using 3,650 days for the averaging time substituted into the above equation, is 3.0 x $10^{-5}$ mg/kg/day. CDIs estimated for dermal absorption of chemicals of potential concern in surface water from Naylors Run are presented in Table 6-19.

Current Land-Use:Direct Contact with Sediments by Children Playing in Naylors Run

Children may be exposed to chemicals of potential concern in sediments in Naylors Run while playing or wading. The estimated exposure to a chemical is based on the amount absorbed through the skin and incidentally ingested. Studies have not been performed specifically on sediment, but much of the information on exposure to soil can be applied to sediments. The following sections describe the two

Table 6-19

Chronic Daily Intakes (CDIs) Estimated for Direct Contact with Surface Water from Naylors Run by Children for the RME Case

| Chemical (a) | RME Exposure Point Concentration (ug/L) | RME CDIs (mg/kg/day) | |
|---|---|---|---|
| | | Carcinogens | Noncarcinogens |
| Organics: | | | |
| Dieldrin | 0.3 | 1.3E-9 | 9.0E-9 |
| Heptachlor Epoxide | 0.8 | 3.4E-9 | 2.4E-8 |
| Benzo(a)pyrene (Equilavent) | 0.3 | 1.3E-9 | 9.0E-9 |
| Pentachlorophenol | 1.200 | 5.2E-6 | 3.6E-5 |
| 2,3,7,8-TCDD (Equivalent) | 3.0E-4 | 1.2E-12 | 8.7E-12 |
| Inorganics: | | | |
| Manganese | 10.100 | --- | 3.0E-4 |
| Thallium | 3.3 | --- | 9.9E-8 |

--- No toxicity criteria available; therefore, a CDI was not estimated.

(a) Toxicity criteria were not available for cobalt and lead; therefore, CDIs were not estimated.

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potential routes of exposure from direct contact with sediments: incidental ingestion of sediments and dermal absorption.

Exposure to Sediments via Ingestion - The ingestion of soil and potentially sediments by children is considered to be a normal phase of childhood development (Baltrop et al. 1963, Robischon 1971, and Ziai 1983). Usually temporary, this behavior may result from normal mouthing, incidental hand-to-mouth activity, and/or dermal absorption (EPA 1989a). Ingestion of soil and sediment past the ages of 6 or 7 has seemingly been termed "abnormal" and is frequently the result of developmental problems (Lourie et al. 1963, Paustenbach et al. 1986). This behavior is otherwise known as pica-abnormal ingestion of a non-food substance (EPA 1989b).

Potential exposures to chemicals of potential concern in sediment via incidental ingestion for the RME case were calculated using the following equation:

$$CDI\ (mg/kg/day) = \frac{(EPC)\ (CF)\ (IR)\ (FI)\ (EF)\ (ED)\ (RBF)}{(BW)\ (AT)}$$

where:

CDI = Chronic Daily Intake (mg/kg/day);
EPC = Exposure Point Concentration (mg/kg for inorganics, ug/kg for organics);
CF = Conversion Factor ($10^{-6}$ kg/mg for inorganics) ($10^{-9}$ kg/ug for organics);
IR = Ingestion Rate (mg/day);
FI = Fraction Ingested from Contaminated Source (unitless);
EF = Exposure Frequency (days/year);
ED = Exposure Duration (years);
RBF = Relative Bioavailability Factor (unitless);
BW = Body Weight (kg); and
AT = Averaging Time (days).

Exposure parameter values used to estimate exposure to children via incidental

ingestion of sediments are discussed below and summarized in Table 6-20.

EPC: The methods for estimating exposure point concentrations are presented in Section 6.1.3.2.

CF: The conversion factor of $10^{-6}$ kg/mg was used to convert mass units for inorganics. The conversion factor of $10^{-9}$ kg/ug was used to convert mass units for organics.

IR: Several studies have been performed to estimate the amount of soil ingested by children. Recent studies performed have used tracer elements in feces and soil to estimate the amount of ingested soil (EPA 1989b). Calabrese et al. (1987) estimated that the average 95th percentile of soil ingestion rates for the three best tracers evaluated was approximately 200 mg/day. Problems with the analytical results for the Calabrese study, however, were found. Binder et al. (1986) used three tracer elements to estimate soil ingestion. The three tracer element results were averaged for an estimated average soil ingestion of 108 mg/day with a range of 100 mg/day to 200 mg/day (EPA 1989b). Van Wijnen et al. (1990) reported that the estimated range of 90th percentiles of ingestion rates ranged from 190 mg/day during normal activities to 300 mg/day duringvacationing at campgrounds. The interim final guidance for soil ingestion rates released by the Office of Soil Waste and Emergency Response (OSWER) recommended using 200 mg/day as an upper-bound soil ingestion rate for children under the age of 6 (EPA 1989d). The 200 mg/day ingestion rate appears to be a reasonable upper-bound value given the supporting research discussed above. A soil ingestion rate of 100 mg/day was recommended for children over the age of 6 and adults (EPA 1989a,d). For the age group evaluated for this pathway (i.e., 2 to 12), a weighted average ingestion rate

Table 6-20

Exposure Parameters Used to Estimate
Exposure to Children via Incidental Ingestion of Sediments in Naylors Run

| Parameter | Value | Reference |
|---|---|---|
| CF | | |
| Organics | $10^{-9}$ kg/mg | |
| Inorganics | $10^{-6}$ kg/mg | |
| IR | 140 mg/day | (EPA, 1989b) |
| FI | 1 | (EPA, 1989a) |
| EF | 125 days/year | (EPA, 1989a) |
| ED | 10 years | (EPA, 1989a) |
| RBF | | |
| Semi-Volatile Organic Compound | .5 | (Poiger and Schlatter, 1980 McConnell et al., 1984, Lucier et al, 1986, Wending et al. 1989, and van den Berg et al., 1986, 1987) |
| Volatile Organics and Inorganics | 1 | Assumed value |
| BW | 25 kg | (EPA, 1985) |
| AT | | |
| Carcinogens | 25550 days | (EPA, 1989a) |
| Non-carcinogens | 3650 | (EPA, 1989a) |

of 140 mg/day was calculated using the EPA (1989a,d) recommended ingestion rates (i.e., 200 mg/day for children between the ages of 2 to 6, and 100 mg/day for children between the ages of 6 to 12).

FI: The fraction ingested from the contaminated source was conservatively assumed to be one (1).

EF: For the exposure frequency, it was assumed that children would play in Naylors Run three times per week for 10 weeks in the spring and fall when the temperature is above freezing (total of 60 days). In the summer months, accounting for warmer weather and schools being closed, children's exposure is considered to be up to five times per week for approximately thirteen weeks. Therefore, the exposure frequency would be 65 days during the summer. The total number of days exposed per year for the RME case was estimated to be 125 days/year (EPA 1989a).

ED: Children were assumed to play in the area between the ages of 2 and 12. Therefore, the exposure duration is 10 years. Children in this age group are more likely to engage in the activity outlined in this pathway than during other ages. In addition, children in this age group may have higher exposure (mg/kg/day) because of their lower body weights (kg) than older children which would have higher body weights.

RBF: The relative bioavailability factor is used to adjust exposure to chemicals of potential concern which tightly bind to a soil/sediment matrix. Many chemicals which adsorb to soil and sediment particles may be less bioavailable than when the chemical is administered in water or oil, which is the typical vehicle used in laboratory toxicity tests. Experimental data on the relative bioavailability

of the chemicals of potential concern are limited. Several studies have been conducted on dioxin which show the relative bioavailability to range from 7% to 50% (Poiger and Schlatter 1980, McConnell at al. 1984, Lucier et al. 1986, Wendling et al. 1989, and Van den Berg et al. 1986, 1987). To be conservative, all semi-volatile organic compounds (e.g., dioxins and furans, pesticides, phthalates, PCP, PCBs, and PAHs) are assumed to have a relative bioavailability factor of 50 percent. Other volatile organic compounds and inorganics are assumed to have a relative bioavailability factor of one (1). This is a conservative assumption which would tend to overestimate the bioavailability for some compounds.

BW: The mean body weight for both male and female children between the ages of 2 to 12 is approximately 25 kg (EPA 1985b).

AT: The averaging time is 10 years (exposure duration) x 365 days/year for noncarcinogens and 70 years (lifetime) x 365 days/year for carcinogens.

An example calculation of the RME CDI for semi-volatile carcinogens assuming an exposure point concentration of 1 ug/kg is presented below:

$$CDI\ (mg/kg/day) = \frac{(1\ ug/kg)(10^{-3}\ kg/ug)(140\ mg/day)(1)(125\ days/year)(10\ years)(.5)}{(25\ kg)(25550\ days)}$$

$$CDI_{carcinogens} = 1.4 \times 10^{-10}\ mg/kg/day$$

For semi-volatile organic compounds (1 ug/kg exposure point concentration), the RME CDI is estimated to be $1.4 \times 10^{-10}$ mg/kg/day and $9.6 \times 10^{-10}$ mg/kg/day, for evaluating carcinogenic and noncarcinogenic effects respectively. For volatile organic compounds (1 ug/kg exposure point concentration), the RME CDI is estimated to be $2.7 \times 10^{-10}$ mg/kg/day and $1.9 \times 10^{-9}$ mg/kg-day, for evaluating carcinogenic and noncarcinogenic effects, respectively. For inorganic compounds (1 mg/kg exposure point concentration), the RME CDI is estimated to be $2.2 \times 10^{-7}$ mg/kg/day and $1.5 \times 10^{-6}$ mg/kg/day, for evaluating carcinogenic and noncarcinogenic effects, respectively. CDIs estimated for incidental ingestion of chemicals of potential concern in sediments from Naylors Run are presented in Table 6-21.

Exposure to Sediments via Dermal Absorption - EPA (1989a) recommends using the soil dermal contact equation for sediment, although due to their textures, most sediments are probably less likely to adhere to the skin than soil. This assessment will focus on the dermal absorption of organic compounds of concern since laboratory studies (Skog and Wahlberg 1964, Wahlberg 1968a,b) have shown that dermal absorption of inorganic compounds bound in a soil/sediment matrix is negligible.

Potential exposures to organic chemicals of potential concern in sediment via dermal absorption for the RME case were calculated using the following equation:

$$CDI\ (mg/kg/day) = \frac{(EPC)\ (CF)\ (SA)\ (AF)\ (ABS)\ (EF)\ (ED)}{(BW)\ (AT)}$$

where:

EPC = Exposure Point Concentration (ug/kg);
CF = Conversion Factor ($10^{-9}$ kg/ug);
SA = Skin Surface Area Available for Contact ($cm^2$/day);
AF = Soil to Skin Adherence Factor ($mg/cm^2$);
ABS = Dermal Absorption Factor (unitless);
EF = Exposure Frequency (days/year);
ED = Exposure Duration (years);
BW = Body Weight (kg); and
AT = Averaging Time (days).

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Table 6-21

Chronic Daily Intakes (CDIs) Estimated for Direct Contact with Sediments Naylors Run for Children Playing in Naylors Run for the RME Case

| Chemical (a) | RME Exposure Point Concentration (Organics: ug/kg Inorganics: mg/kg), | RME CDIs for Incidential Ingestion (mg/kg/day) (b) | | RME CDIs for Dermal Absorption (mg/kg/day) (b) | |
|---|---|---|---|---|---|
| | | Carcinogens | Noncarcinogens | Carcinogens | Noncarcinogens |
| Organics: | | | | | |
| Benzo(a)pyrene (Equivalent) | 28,000.0 | 3.9E-6 | --- | 3.9E-6 | --- |
| Chlordane (total) | 230.0 | 3.2E-8 | 2.2E-7 | 3.2E-8 | 2.3E-7 |
| Fluoranthene | 21,000.0 | --- | 2.0E-5 | --- | 2.1E-5 |
| Pentachlorophenol | 3,000.0 | 4.2E-7 | 2.9E-6 | 4.2E-7 | 3.0E-6 |
| 2,3,7,8-TCDD (Equivalent) | 0.12 | 1.7E-11 | 1.2E-10 | 1.7E-11 | 1.2E-10 |
| Inorganics (b): | | | | | |
| Antimony | 14.1 | --- | 2.1E-5 | | |
| Arsenic | 37.6 | 8.3E-6 | 5.6E-5 | | |
| Barium | 415.0 | --- | 6.2E-4 | | |
| Chromium | 532.0 | --- | 8.0E-4 | | |
| Manganese | 4,750.0 | --- | 7.1E-3 | | |
| Nickel | 33.0 | --- | 5.0E-5 | | |
| Thallium | 1.0 | --- | 1.5E-6 | | |
| Vanadium | 118.0 | --- | 1.8E-4 | | |

--- No toxicity criteria available; therefore, a CDI was not estimated.

(a) Toxicity criteria were not available for dibenzofuran, endosulfan sulfate, acenaphthene, phenanthrene, aluminum, cobalt, and lead; therefore, CDIs were not estimated.

(b) Dermal absorption of inorganic chemicals was assumed to be zero.

Exposure parameter values used to estimate exposure to children via dermal absorption of chemicals in sediments are discussed below and summarized in Table 6-22.

EPC: The methods for estimating exposure point concentrations are presented in Section 6.1.3.2.

CF: The conversion factor of $10^{-9}$ kg/ug is used to convert mass units.

SA: Approximately one-third of the total surface area of the hands, arms, and legs were assumed to directly contact sediments. Thus, approximately 1000 $cm^2$ of the body surface would contact contaminated sediments based on data presented in EPA (1985a, 1989c) for children ages 2 to 12. The 50th percentile of the surface area of the hands, arms, and legs was used, rather than an upper-bound percentile, because it reflects the best estimate of the surface area for the individual with the 50th percentile body weight (EPA 1989a).

AF: A skin-to-soil adherence factor of 1.45 mg/cm has been calculated based commercial potting soil (EPA 1989a).

ABS: The absorption factor reflects the percentage of a chemical that contacts the skin which will pass through the skin to the stratum corneum and into the bloodstream. Factors influencing dermal absorption from a soil or sediment matrix include the affinity of the compound for the soil matrix, the presence of other agents that might facilitate the permeability of a chemical, as well as the properties of the skin itself (EPA 1988a). Based on results from Yang et al. (1986a,b), Wester et al. (1987), and Poiger and Schlatter (1980), it is assumed that 5 percent of the semi-volatile compounds (e.g., dioxins and furans, PAHs, PCP, phthalates, pesticides, and PCBs) in sediment are absorbed through the skin.

Table 6-22
Exposure Parameters Used to Estimate
Exposure to Children via Dermal Absorption of Chemicals in Sediments from Naylors Run

| Parameter | Value | Reference |
|---|---|---|
| CF | $10^{-9}$ kg/ug | |
| SA | 1000 $cm^2$/day | (EPA, 1989a) |
| AF | 1.45 $mg/cm^2$ | (EPA, 1989a) |
| ABS | | (EPA, 1989a) |
| Semi-Volatile Organic Compounds | .05 | (Yang et al., 1986a,b Wester et al., 1987, |
| Volatile Organic Compounds | .1 | Poiger & Schlatter, 1980) |
| Inorganics | 0 | (Skog & Wahlberg, 1964, Wahlberg, 1968a,b) |
| EF | 125 days/year | (EPA, 1989a) |
| ED | 10 years | (EPA, 1989a) |
| BW | 25 kg | (EPA, 1985) |
| AT | | |
| Carcinogens | 25550 days | (EPA, 1989a) |
| Noncarcinogens | 3650 days | (EPA, 1989a) |

There is insufficient experimental evidence for deriving dermal absorption factors for other organic chemicals of potential concern. Therefore, considering the relative absorptive properties of these chemicals compared to those with known values, it is conservatively assumed that 10 percent is absorbed through the skin to the bloodstream. Based on laboratory studies (Skog and Wahlberg 1964, Wahlberg 1968a,b), inorganic compounds are not considered to be absorbed and thus exposure to inorganics from dermal contact is assumed to be zero.

EF: For the exposure frequency, it was assumed that children would play in Naylors Run three times per week for 10 weeks in the spring and fall when the temperature is above freezing (total of 60 days). In the summer months, accounting for warmer weather and schools being closed, children's exposure is considered to be up to five times per week for approximately thirteen weeks. Therefore, the exposure frequency would be 65 days during the summer. The total number of days exposed per year for the RME case was estimated to be 125 days/year (EPA 1989a).

ED: Children were assumed to play in the area between the ages of 2 and 12. Therefore, the exposure duration is 10 years. Children in this age group are more likely to engage in the activity outlined in this pathway than during other ages. In addition, children in this age group may have higher exposure (mg/kg/day) because of their lower body weights (kg) than older children which would have higher body weights.

BW: The mean body weight for both male and female children between the ages of 2 to 12 was approximately 25 kg (EPA 1985a).

AT: The averaging time is 10 years (exposure duration) x 365 days/year for noncarcinogens and 70 years (lifetime) x 365 days/year for carcinogens.

An example calculation of the RME CDI for semi-volatile carcinogens assuming an exposure point concentration of 1 ug/kg is presented below:

$$CDI = \frac{(1\ ug/kg)(10^{-9}\ kg/ug)(1000\ cm^2/day)(1.45\ mg/cm^2)(.05)(125\ days/year)(10\ years)}{(25\ kg)(25550\ days)}$$

$$CDI_{carcinogens} = 1.4 \times 10^{-10}\ mg/kg/day$$

For semi-volatile organic compounds (1 ug/kg exposure point concentration), the RME CDI is estimated to be 1.4 x $10^{-10}$ mg/kg/day and 9.9 x $10^{-10}$ mg/kg/day, for evaluating carcinogenic and noncarcinogenic effects respectively. For volatile organic compounds (1 ug/kg exposure point concentration), the RME CDI is estimated to be 2.7 x $10^{-10}$ mg/kg/day and 2.0 x $10^{-9}$ mg/kg-day, for evaluating carcinogenic and noncarcinogenic effects, respectively. CDIs estimated for dermal absorption of chemicals of potential concern in sediments from Naylors Run are presented in Table 6-21.

Current Land-Use: Ingestion of Fish from Cobbs Creek

Recreational fisherman who fish along Cobbs Creek may be exposed to chemicals of potential concern from the consumption of contaminated fish tissue. EPA (1989d) guidance entitled "Assessing Human Health Risk from Chemically Contaminated Fish and Shellfish" was used to estimate exposure from ingestion of fish. The quantity and rate of fish consumption will vary depending on the region of the country, age group, fishing pattern, and race. The following estimates

concentrate on the subpopulation of recreational fishermen and their families.

Potential exposures to recreational fisherman via ingestion of contaminated fish for the RME case were calculated using the following equation:

$$CDI = \frac{(EPC)(CF_1)(CF_2)(IR)(FI)(EF)(ED)}{(BW)(AT)}$$

where:

CDI = Chronic Daily Intake (mg/kg/day);
EPC = Exposure Point Concentration (ug/kg);
$CF_1$ = Conversion Factor ($10^{-9}$ kg/ug);
$CF_2$ = Conversion Factor ($10^{-3}$ mg/g);
IR = Ingestion Rate (g/day);
FI = Fraction Ingested from Contaminated Source (unitless);
EF = Exposure Frequency (days/year);
ED = Exposure Duration (years);
BW = Body Weight (kg); and
AT = Averaging Time (days).

Exposure parameter values used to estimate exposure to recreational fisherman via ingestion of fish from Cobbs Creek are discussed below and summarized in Table 6-23.

EPC: The methods for estimating exposure point concentrations are presented in Section 6.1.3.2.

$CF_1$: This conversion factor of $10^{-9}$ kg/ug is used to convert fish concentration mass units.

$CF_2$: A second conversion factor of $10^{-3}$ mg/g is used to convert the fish ingestion rate mass units.

IR: Pao et al. (1982) estimated that 132 g/day represented the 95th percentile for individuals consuming fin fish averaged over a three day period. Pao et al. (1982) estimated that 38 g/day represented

Table 6-23

Exposure Parameter Values Used to Estimate
Exposure to Recreational Fisherman from Ingestion of Fish from Cobbs Creek

| Parameter | Value | Reference |
|---|---|---|
| $CF_1$ | $10^{-3}$ mg/g | - - - |
| $CF_2$ | $10^{-9}$ kg/ug | - - - |
| IR | 41.7 g/day | (SRI, 1980) |
| FI | 1 | (EPA, 1989a) |
| EF | 365 days/year | (EPA, 1980) |
| ED | 30 years | (EPA, 1989a) |
| BW | 70 kg | (EPA, 1989a) |
| AT | | |
| Carcinogens | 25,550 days | (EPA, 1989a) |
| Non-carcinogens | 10,950 days | (EPA, 1989a) |

the 50th percentile for the consumption of fin fish averaged over a three day period. SRI (1980) reported that the daily average 95th percentile for fish ingestion was 41.7 g/day. The value reported by SRI (1980) of 41.7 g/day was used for the RME case in this assessment.

FI: This value is a measure of the fraction of fish ingested from Cobbs Creek. To be conservative, 100 percent (FI=1) of the non-commercial fish ingested was assumed to come from Cobbs Creek.

EF: An exposure frequency of 365 days/year was used since the ingestion rate is based on an annual average 95th percentile.

ED: The 90th percentile of the number of years an individual lives in the same area (i.e., 30 years) was used as the exposure duration (EPA 1989a).

BW: EPA (1985a) calculated an average body weight of 71.8 kg. This value is approximately equal to the consensus value of 70kg which is generally used as the average body weight.

AT: The averaging time is 30 years (exposure duration) x 365 days/year for noncarcinogens and 70 years (lifetime) x 365 days/year for carcinogens.

An example calculation of the RME CDI for chemicals of potential concern for ingestion of fish from Cobbs Creek assuming an exposure point concentration of 1 ug/kg is presented below:

$$CDI_{mg/kg/day} = \frac{(1\ ug/kg)(10^{-6}\ kg/ug)(10^{3}\ mg/g)(41.7\ g/day)(1)(365\ days/year)(30\ years)}{(70\ kg)(25550\ days)}$$

AR300618

$$CDI_{mg/kg-day} = 2.6 \times 10^{-7}$$

Thus, the CDI for ingestion of fish for carcinogens is 2.6 x $10^{-7}$ mg/kg/day assuming a 1 ug/kg exposure point concentration in fish tissue. The CDI for ingestion of fish for noncarcinogens is 6.0 x $10^{-7}$ mg/kg/day. CDIs estimated for ingestion of fish for chemicals of potential concern detected in fish tissue collected from Cobbs Creek are presented in Table 6-24.

Future Land-Use: Ingestion of Chemicals in Groundwater

Chemicals of potential concern in groundwater may be ingested if groundwater is used as a source of drinking water under future land-use of the site. It is assumed that a resident may install a well in the vicinity of the most contaminated monitoring wells at the site. It should be emphasized that it is highly unlikely that residents would actually use groundwater in the vicinity of the Havertown PCP site as a source of drinking water in the future. Residents in the area currently use municipal water provided by the City of Havertown, and residential homes constructed in the future would likely be hooked-up to the city water supply system. In addition, further commercial and industrial development would likely use water supplied by the City of Havertown. However, this pathway was quantitatively evaluated in this report in order to justify further restrictions of groundwater use and in order to provide the basis for making risk management decisions concerning remediation of groundwater at the Havertown PCP site.

Potential exposures to chemicals of potential concern via ingestion of groundwater for the RME case were calculated using the following equation:

$$CDI(mg/kg/day) = \frac{(EPC)\ (IR)\ (EF)\ (ED)\ (CF)}{(BW)\ (AT)}$$

Table 6-24

Chronic Daily Intakes (CDIs) Estimated for
Ingestion of Fish Caught Downstream
from the Havertown PCP Site in Cobbs Creek

| Chemical | RME Exposure Point Concentration (Units: ug/kg) (a) | RME CDI (a) | |
|---|---|---|---|
| | | Carcinogens | Noncarcinogens |
| Chlordane (total) | 238 | 6.1E-5 | 1.4E-4 |
| Dieldrin | 450 | 1.2E-4 | 2.7E-4 |
| Heptachlor Epoxide | 37 | 9.5E-6 | 2.2E-5 |
| 2,3,7,8-TCDD (Equivalent) | 0.007 | 3E-9 | 4.2E-9 |

(a) Exposure point concentration and exposure associated with ingestion of white suckers. Exposure associated with ingestion of sport fish may be much lower given their foraging behavior which may result in lower fish tissue concentrations.

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where:

CDI = Chronic Daily Intake (mg/kg/day);
EPC = Exposure Point Concentration (ug/L);
CF = $10^{-3}$ mg/ug;
IR = Ingestion Rate (L/day);
EF = Exposure Frequency (days/year);
ED = Exposure Duration (years);
BW = Body Weight (kg); and
AT = Averaging Time (days).

Exposure parameter values used to estimate exposure to hypothetical residents via ingestion of groundwater are discussed below and summarized in Table 6-25.

EPC: The methods for estimating exposure point concentrations are presented in Section 6.1.3.2.

CF: A conversion factor of $10^{-3}$ mg/ug was used to convert mass units.

IR: Gillies and Paulin (1983) estimated the 90th percentile of daily water consumption to be 1.9 L/day. Studies conducted by Cantor etal. (1987) suggested an ingestion rate of 2.0 L/day represented a 90th percentile of the ingestion rate distribution. EPA (1989a), after reviewing available data, concluded that a groundwater ingestion rate of 2.0 L/day represents a reasonable maximum ingestion rate. Using this value in the risk assessment, however, assumes that the individual ingests water only from one's own tap during the course of the day. Data presented in EPA (1989b) suggest that individuals may receive approximately 30 percent of their drinking water from sources other than their own well.

EF: For the RME it is assumed that a resident ingests groundwater from their own private well 365 days per year.

ED: The 90th percentile of the number of years an individual lives in

Table 6-25

Exposure Parameter Values used to Estimate
Exposure to Hypothetical Residents via Ingestion of Groundwater

| Parameter | Value | Reference |
|---|---|---|
| CF | $10^3$ mg/ug | - - - |
| IR | 2 L/day | (EPA, 1985) |
| EF | 365 days/year | (EPA, 1989a) |
| ED | 30 years | (EPA, 1989a) |
| BW | 70 kg | (EPA, 1985) |
| AT | | |
| Carcinogens | 25,550 days | (EPA, 1989a) |
| Non-carcinogens | 10,950 days | (EPA, 1989a) |

AR300622

the same area (i.e., 30 years) was used as the exposure duration (EPA 1989a).

BW: EPA (1985a) calculated an average body weight for males and females of 71.8 kg. This value is approximately equal to the consensus value of 70 kg which is typically used as the average body weight.

AT: The averaging time is 30 years (exposure duration ) x 365 days/year for noncarcinogens and 70 years (lifetime) x 365 days/year for carcinogens.

An example calculation of the RME CDI for chemicals of potential concern for ingestion of groundwater assuming an exposure point concentration of 1 ug/L is presented below:

$$CDI_{mg/kg/day} = \frac{(1\ ug/L)(1 \times 10^{-3}\ mg/ug)(2\ L/day)(365\ days/year)(30\ years)}{(70\ kg)(25,550\ days)}$$

$$CDI = 1.2 \times 10^{-4}\ mg/kg/day$$

Thus, the CDI for ingestion of groundwater for carcinogens is $1.2 \times 10^{-5}$ mg/kg/day assuming a chemical concentration of 1 ug/L. The CDI for ingestion of groundwater for noncarcinogens is $2.9 \times 10^{-5}$ mg/kg/day. CDIs estimated for ingestion of groundwater for chemicals of potential concern are presented in Table 6-26.

Future Land-Use: Inhalation of VOCs while Showering

There is research evidence to suggest that the exposure to VOCs via inhalation while showering is approximately equal to the exposure from ingestion. Using the exposure calculated for ingestion in place of the inhalation exposure would be practical given the level-of-effort necessary for performing the shower model for each chemical of potential concern. Certain EPA Regions such as Region IX have adopted this approach as a standard practice for estimating exposure and risk via

Table 6-26

Chronic Daily Intakes (CDIs) Estimated for Ingestion of Groundwater from the Havertown PCP Site by Hypothetical Residents for the RME Case

| Chemical (a) | RME Exposure Point Concentration (ug/L) | RME CDIs (mg/kg/day) | |
|---|---|---|---|
| | | Carcinogens | Noncarcinogens |
| Organics: | | | |
| Benzene | 230 | 2.8E-3 | 6.6E-3 |
| 1,2-Dichloroethene (total) | 245 | - - | 7.1E-3 |
| bis(2-Ethylhexyl)phthalate | 180 | 2.2E-3 | 5.2E-3 |
| Benzo(a)pyrene (Equivalent) | 741.9 | 8.9E-3 | 2.2E-2 |
| Fluoranthene | 810 | - - | 2.3E-2 |
| Naphthalene | 24,000 | - - | 6.8E-1 |
| Pentachlorophenol | 80,000 | 9.6E-1 | 2.3E+0 |
| Trichloroethene | 465 | 5.6E-3 | 1.3E-2 |
| Vinyl Chloride | 9.1 | 1.1E-4 | 2.6E-4 |
| 2,3,7,8-TCDD (Equivalent) | 0.174 | 2.1E-6 | 5.0E-6 |
| Inorganics: | | | |
| Arsenic | 22.7 | 2.7E-4 | 6.5E-4 |
| Manganese | 22,600 | - - | 6.4E-1 |
| Thallium | 1.7 | - - | 4.9E-5 |

(a) Toxicity criteria were not available for dibenzofuran, 2-methylnaphthalene, acenapthene, phenanthrene, aluminum, and cobalt; therefore, CDIs were not estimated.

(b) The same CDI is used for evaluating both noncarcinogenic and carcinogenic effects since the individual is assumed to be exposed over a lifetime (see text for further discussion).

inhalation in order to expedite the risk assessment process. A sensitivity analysis was performed to determine whether it is appropriate to use an ingestion rate CDI to estimate exposure from inhalation.

Potential exposure to an individual per shower via inhalation of VOCs for the RME case can be calculated using the following equation (Foster and Chrostowski 1987):

$$Einh = \frac{(Vr)(S)}{(R)(10^6)}(D_s + \exp(-RD_t)/R - \exp[R(D_s - D_t)]/R)$$

where:

Einh = Inhalation Exposure per Shower (mg/kg-shower);
VR = Ventilation Rate (1/min);
BW = Body Weight (kg);
$D_t$ = Total Duration in Shower Room (min);
$D_s$ = Shower Duration (min);
S = Indoor VOC Generation Rate (ug/$m^3$-min); and
R = Air Exchange Rate ($min^{-1}$)

The model developed by Foster and Chrostowski (1987) has been validated based on available experimental data. The results of the validation indicate that the model produces reliable air concentrations from the volatilization component. Exposure per shower calculated from the model can be used in the following equation to estimate the CDI.

$$CDI_{mg/kg/day} = \frac{(EF)(ED)(Einh)}{(AT)}$$

where:

CDI = Chronic Daily Intake (mg/kg/day);
EF = Exposure Frequency (shower/year);
ED = Exposure Duration (years);
Einh = Inhalation Exposure per Shower (mg/kg/shower); and
AT = Averaging Time (days).

AR300625

Table 6-27 presents CDIs estimated for five VOCs in shower room air modeled using the approach outlined by Foster and Chrostowski (1987) and CDIs estimated for ingestion of groundwater. In comparing exposures from daily groundwater ingestion to exposures from inhalation during daily showering, it may be concluded that these pathways presented similar exposures. Therefore, it is reasonable to assume that VOC exposures to individuals via inhalation are equivalent to exposures from ingestion. Thus, groundwater ingestion exposures calculated for VOCs presented in Table 6-26 will be used as the CDIs for inhalation exposure in this assessment. However, inhalation toxicity criteria will be used, where available, for estimating potential carcinogenic and noncarcinogenic risks.

Estimating Exposure to Lead using Pharmacokinetic Modeling

A pharmacokinetic model was used to estimate exposure to children from lead present in Naylors Run (all of the lead data in groundwater was rejected due to blank contamination). The Integrated Uptake/Biokinetic (IU/BK) model is a computerized pharmacokinetic model that analyzes the affects of lead upon chemicals (i.e., estimating CDIs) cannot be used to analyze the effects of lead poisoning because unlike most chemicals (which have a threshold for noncarcinogenic effects) lead may impact development of neurological function at any dose level (i.e., no threshold).

The IU/BK model essentially quantifies the distribution of possible lead concentrations in the blood using a multimedia approach. The IU/BK consists of two basic modules: 1) the uptake of lead, and 2) the biokinetics of lead in the body. Uptake of lead is defined as the amount of lead that is absorbed into the body's blood-plasma system from various sources (i.e, ingestion, inhalation, and dermal absorption). Using absorption factors calculated from the above uptakes, the biokinetic model calculates the amount of lead that will occur in a number

Table 6-27
Comparison of Exposures Estimated for
Inhalation While Showering versus Ingestion

| Chemical | Exposure from Inhalation During Shower (mg/kg/day)[a] | Exposure from Ingestion of Groundwater (mg/kg/day)[b] |
|---|---|---|
| Benzene | $2.26 \times 10^{-3}$ | $2.18 \times 10^{-3}$ |
| Chloroform | $1.88 \times 10^{-3}$ | $2.18 \times 10^{-3}$ |
| Tetrachloroethane | $1.75 \times 10^{-3}$ | $2.18 \times 10^{-3}$ |
| Trichloroethene | $1.89 \times 10^{-3}$ | $2.18 \times 10^{-3}$ |
| Vinyl chloride | $2.50 \times 10^{-3}$ | $2.18 \times 10^{-3}$ |

(a) The upper-bound scenario for inhalation during a shower using a water concentration = 75 ug/L, an air exchange rate = 0.5 $hr^{-1}$, and a 15-minute shower with 5 minutes in the shower room after the water was turned off.

(b) An exposure point concentration of 75 ug/L was used for all chemicals (see also the discussion on estimating exposure for ingestion pathways).

of "body compartments". In the body, lead is exchanged among body compartments such as plasma and the extra cellular fluid (ECF) pool, red blood cells (RBC pool), kidneys, liver, trabecular bone, cortical bone, and other soft tissue pools. The important factor of the biokinetic module is the transition time for the movement of lead between compartments (which include removal by feces and urine). The transition time is the rate determining factor which determines the rate at which lead enters, resides, and then leaves each compartment during a monthly iteration. The transition time is calculated on a monthly basis and is dependant upon the body weight and individual compartment weight at that monthly age.

In this assessment, potential exposure to lead via incidental ingestion of sediments from Naylors Run were evaluated. The exposure point concentration for lead in sediments was used as the soil concentration in the IU/BK model. Default values for other parameter values in the model were used to estimate exposure from other sources such as drinking water, air, maternal sources, etc.

In accordance with EPA Region III guidance (EPA 1991c), the default geometric standard deviation (GSD) of 1.42 was changed to 1.7, based on more recent data on the GSD of blood lead levels in children at hazardous waste sites (i.e., Baltimore Lead Abatement and Cincinnati Lead Abatement studies, as cited in EPA Region III guidance [1991c]).

Estimating Exposure to Nursing Infants using Pharmacokinetic Modeling

Nursing infants may be indirectly exposed to dioxins and furans in fish tissue (under current land-use conditions) and groundwater (under future land-use conditions) via lactational transfer assuming that the mother is directly exposed to dioxins and furans in fish tissue or groundwater. Exposure to nursing infants was estimated using a pharmacokinetic model that quantifies exposure to the infant based on exposure to the mother. This model assumes that prior to

lactation, the levels of dioxin and furan have reached steady-state conditions in the fat tissue of the mother. Thus, the mother has been exposed for several years prior to the commencement of breast feeding.

Modeling Approach - The pharmacokinetic model used in this report to estimate exposure to nursing infants was derived from modeling results presented in Smith (1987). Smith (1987) derived the following equation for estimating the dose (mg) to the nursing infant on day "T" after the commencement of breast feeding:

$$Dose(mg) = \frac{bf_1f_3f_4}{f_2}\left[me^{k_bT}\left(\frac{1}{k} - \frac{1}{k_b}\right) + \frac{m}{k_b}\right]$$

where:

| | | |
|---|---|---|
| b | = | average breast-milk ingested per day by the infant (kg); |
| $f_1$ | = | proportion of maternal dioxin and furan in fat; |
| $f_2$ | = | proportion of maternal weight that is fat; |
| $f_3$ | = | proportion of breast-milk that is fat; |
| $f_4$ | = | proportion of ingested dioxin and furan that is absorbed; |
| m | = | exposure to the mother (mg/kg/day); |
| T | = | day T of nursing; |

$$K_b = \frac{\ln(2)}{h} + \frac{bf_1f_3}{f_2W}$$

h = half-life of dioxin

$$k = \frac{\ln(2)}{h}$$ ;and

W = maternal body weight (kg).

This equation takes into account the release of dioxin and furan from the body due to lactational transfer. The highest dose is received by the infant on the first day of feeding, followed by lower doses after subsequent feedings. To estimate the total dose (mg) to the child over the entire exposure duration (i.e., 2 years), the sum of all the daily doses is calculated using the equation

presented above. To estimate the CDI for evaluating noncarcinogenic effects, the sum of the daily doses is divided by the number of days in the exposure duration and average body weight of the infant. To estimate the CDI for evaluating the increased risk of carcinogenic effects, the sum of the daily intakes is divided by an average life-time and average body weight of the infant.

The following equation was used to estimate the CDI of a nursing infant:

$$\textit{Chronic Daily Intake (mg/kg/day)} = \frac{bf_1f_3f_4}{f_2W_{ED/2}AT}\sum_{i=1}^{ED}\left[me^{k_bT}\left(\frac{1}{k}-\frac{1}{k_b}\right)+\frac{m}{k_b}\right]$$

where (also see descriptions above):

| | | |
|---|---|---|
| $W_{ED/2}$ | = | body weight of the infant at one-half the exposure duration; |
| ED | = | exposure duration (days); and |
| AT | = | averaging time, 730 days (i.e., numbers of days in the exposure duration). |

The above equation can be solved directly using the equation presented below, since the above equation represents the summation of a finite geometric series (see descriptions above for model parameters).

$$\textit{Chronic Daily Intake (mg/kg/day)} = \frac{bf_1f_3f_4m}{f_2W_{ED/2}AT}\left[\left(\frac{1}{k}-\frac{1}{k_b}\right)\frac{(1-e^{-k_bED})}{(1-e^{-k_b})}+\frac{ED}{k_b}\right]$$

To calculate the CDI for evaluating carcinogenic risk, the averaging time is set equal to the number of days in a life-time.

Modeling Assumptions - Parameter values used to estimate exposure to nursing infants are presented in Table 6-28. The parameter values represent the best

Table 6-28

Parameter Values Used to Estimate Exposure to Nursing Infants

| Exposure Parameter | Value | Description | Reference |
|---|---|---|---|
| b | 0.8 kg/day | Kilograms of breast milk ingested by the infant per day | Butte et al. (1984) Whitehead and Paul (1981) (as cited in Smith, 1987) |
| $f_1$ | 0.51(a) | Proportion of maternal dioxin and furan in fat | King at al. (1983) |
| $f_2$ | 0.3 | Proportion of maternal weight that is fat | Timson and Coffman (1984) Butte et al. (1984) |
| $f_3$ | 0.036 | Proportion of breast-milk that is fat | EPA (1988a) |
| $f_4$ | 0.68 | Proportion of dioxin and furan absorbed | EPA (1988a) |
| m | Pathway specific | Maternal Exposure to Dioxin Equivalents | |
| n | 1.825 days (i.e., 5 years) | Half-life of dioxin equivalents | EPA (1988a) |
| k | 3.8E-4(b) | Elimination rate constanc (c) | EPA (1988a) |
| $k_b$ | 1.1E-3(d) | Adjusted Elimination rate constant (e) | Smith (1987) |
| W | 70 kg | Maternal weight | EPA (1988a) |
| $W_{ED/2}$ | 8.3 | Infant body weight at one year (i.e., halt the exposure duration) | EPA (1988a) |
| ED | 730 days (i.e., 2 years) | Duration of lactation | |
| AT | 1,825 days (i.e., 2 years) | Averaging time for evaluating noncarcinogenic effects | EPA (1988a) |
| | 27,375 days (i.e., 75 years) | Averaging time for evaluating carcinogenic effects | EPA (1988a) |

(a) Based on the fat volume distribution (12L) divided by the overall volume distribution (23.55L) for dioxin toxicity equivalents in experimental monkeys.

(b) Calculated from the expression $k_e$ = LN (2)/(half-life) and assuming a half-life of 1,825 days (i.e., years).

(c) Rate constant does not factor in maternal losses of dioxin equivalents via breast milk release.

(d)
$$k_b = \frac{0.693}{h} + \frac{bf_1f_3}{f_2W}$$

(e) Rate constant factors in maternal losses via breast milk release.

AR300631

estimates based on existing information. An 8.3 kg nursing infant was assumed to ingest 0.8 kg of breast milk per day over a 2 year lactation period. The mothers breast-milk was assumed to contain 3.6 percent fat and the mothers body was assumed to be 30 percent adipose tissue. The half-life of 2,3,7,8-TCDD (Equivalent) was assumed to be five years. The levels of dioxin and furan in maternal adipose tissue were assumed to have reached steady-state conditions. Dioxin and furan specific model parameters include a fat partitioning coefficient ($f_1$) and percent bioavailability ($f_4$) (i.e., absorption of dioxin through the gastrointestinal tract of the mother). Limited data were available for estimating $f_1$ and $f_4$ parameter values for 2,3,7,8-TCDD (Equivalent). For 2,3,7,8-TCDD (Equivalent), a fat partitioning coefficient of 0.51 ($f_1$) was derived by a study conducted by King et al. (1983), which was cited in EPA (1988a). Fries and Marrow (1975) reported that the percent absorption of 2,3,7,8-TCDD may range from 50% to 60% for rats fed feed. Rose et al. (1976) reported a percent absorption of 86% for 2,3,7,8-TCDD fed acetone and corn oil via gavage. Based on these studies, EPA (1988a) recommends using a 68% absorption factor ($f_4$) for 2,3,7,8-TCDD (Equivalent).

CDIs for nursing infants indirectly exposed to dioxin and furan in fish tissue and groundwater are presented in Table 6-29. For evaluating noncarcinogenic risk, the CDI (mg/kg/day) of 2,3,7,8-TCDD (Equivalent) for nursing infants was estimated to be 8.4 times the maternal CDI over a two year exposure duration. Thus, the noncarcinogenic exposure and risk to nursing infants can be estimated by multiplying the maternal noncarcinogenic exposure by a factor of 8.4. For evaluating carcinogenic risk, the CDI (mg/kg/day) to nursing infants was estimated to be 22 percent of the CDI for the mother, assuming that the infant is exposed only during the first two years of life, while the mother is exposed over a lifetime. Thus, nursing infant exposure may increase an individuals lifetime cancer risk by 22 percent, assuming that the individual is exposed at a similar level as the mother over a lifetime.

Table 6-29

Chronic Daily Intakes (CDIs) Estimated for
Nursing Infants Exposed to 2,3,7,8-TCDD (Equivalent)
via Ingestion of Contaminated Breast Milk

| Maternal Exposure Pathway (a) | Maternal CDI (mg/kg/day) | Nursing Infant CDI | |
|---|---|---|---|
| | | Carcinogens (b) | Noncarcinogens (c) |
| Current Land-Use: | | | |
| Ingestion of fish | 4.2E-9 | 8.4E-10 | 3.3E-8 |
| Future Land-Use: | | | |
| Ingestion of groundwater | 5.0E-6 | 1.0E-6 | 3.9E-5 |

(a) Pathway by which mother is exposed.
(b) Cancer CDI assumes infant is only exposed from ingestion of breast milk. Exposure from other routes after lactation period is assumed to be zero. Infant life-time average exposure (over the 2 years of exposure) is 20 percent of the mother's average lifetime daily exposure.
(c) Infant average daily exposure is approximately 8 times the mother's average daily exposure.

### 6.1.4 Toxicity Assessment

This section evaluates the carcinogenic and noncarcinogenic toxicity of chemicals of potential concern selected in Section 6.1.2. Toxicity assessment is the process of evaluating the potential for a chemical to cause an adverse health effect in humans and, if possible, to quantify the relationship between exposure levels (i.e., dose) and the adverse health effect. Hazard identification is the first step in conducting a toxicity assessment which involves evaluating the potential for a chemical to cause an adverse health effect. Dose-response evaluation is the second step in the toxicity assessment process which attempts to quantify the relationship between dose of the administered chemical and the increased incidence of the adverse health effect.

The slope factor is used to evaluate the potential carcinogenic risks associated with exposure to a chemical of potential concern. The reference dose (i.e., RfD) is used to evaluate the potential noncarcinogenic risks associated with exposure to a chemical of potential concern. Toxicity criteria and supporting toxicity data used in the baseline risk assessment were obtained from the Integrated Risk Information System (IRIS) (EPA 1991d), Fourth Quarter Health Effects Assessment Summary Tables (HEAST) (EPA 1990c), Health Effects Assessment documents, Toxicity Profiles developed by the Agency for Toxic Substances and Disease Registry (ATSDR), and other sources. This report evaluates both chronic oral exposure for all chemicals of potential concern and inhalation exposure for VOCs in groundwater. In addition, dermal absorption of chemicals of potential concern in sediment and surface water were evaluated, however, dermal absorption RfDs and slope factors were not available for the chemicals evaluated in this report. Therefore, oral toxicity criteria were used to evaluate the toxicity of chemicals for the dermal absorption route.

6.1.4.1 Toxicity Criteria for Evaluating Potential Carcinogenic Effects

The slope factor, expressed in $mg/kg/day^{-1}$, quantifies the potential cancer potency of a chemical for evaluating the carcinogenic risks associated with exposure. Unlike noncarcinogenic effects, a small number of molecular events may alter a cell in such a way as to cause uncontrolled cellular proliferation, thereby resulting in disease (i.e., carcinogenic effect). Therefore, any exposure may result in the manifestation of a carcinogenic effect. Thus, no exposure is considered risk free.

To evaluate the potential carcinogenic toxicity of a chemical, EPA first determines the likelihood that the chemical is a human carcinogen. EPA uses a classification system (i.e., weight-of-evidence classification) for characterizing the potential carcinogenicity of a chemical based on the evidence presented in animal and human studies. The weight-of-evidence classification scheme is presented below:

- A - Human Carcinogen;
- B1 - Probable Human Carcinogen, based on limited human data;
- B2 - Probable Human Carcinogen, based on sufficient evidence in animals and inadequate or no evidence in humans;
- C - Possible Human Carcinogen;
- D - Not classifiable as to human carcinogenicity; and
- E - Evidence of noncarcinogenicity for humans.

If the chemical is a human carcinogen (Group A) or a probable human carcinogen (Group B1 or Group B2), then a slope factor is calculated for the chemical which quantifies its cancer potency. In certain cases, slope factors are derived for possible human carcinogens (Group C compounds). Slope factors are derived by extrapolating dose-response relationships measured under high dose conditions in laboratory animal studies or epidemiological studies to low dose conditions typically encountered at Superfund sites. The first step in deriving a slope

factor involves fitting a mathematical model to the experimental data (EPA 1986a). Of the available low dose extrapolation models (i.e., Weibull, probit, logit, one-hit, and gamma multihit models), the more conservative linearized multistage model is typically used to derive a slope factor from animal data. This model assumes that the dose-response relationship at low doses is linear. Once the data are fit using the linearized multistage model, the 95th upper confidence limit on the slope of the line is calculated which represents the slope factor. Slope factors are then verified and validated by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) Workgroup before being placed on IRIS. Slope factors based on epidemiological data are fit on an ad hoc basis. Slope factors and supporting toxicity data for chemicals of potential concern are summarized in Table 6-30.

6.1.4.2 Toxicity Criteria for Evaluating Potential Noncarcinogenic Effects

The reference dose, expressed in mg/kg/day, is used to evaluate the potential noncarcinogenic risks associated with exposure to a chemical of potential concern at a Superfund site. A chronic RfD is defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime based on an administered dose (EPA 1989a). It is assumed that a protective mechanism in the body must be overcome in order for a noncarcinogenic effect to occur (i.e., threshold effect). For example, numerous cells in an organ must be damaged before an effect may be manifested.

In general, RfDs are derived from animal laboratory studies or human epidemiology studies. These studies are reviewed to derive a no-observable-adverse-effect level (NOAEL) for the chemical. The lowest-observable-adverse-effect level (LOAEL) is used when a NOAEL cannot be derived from the study. In this case, an additional uncertainty factor is applied to estimate the RfD. Uncertainty factors (UF) are applied to the NOAEL (or LOAEL) to account for various types of uncertainty including:

Table 6-30

Chronic Carcinogenic Toxicity Criteria (SFs)
for Chemicals of Concern at the Havertown PCP Site

| Route/Chemical (a) | Slope Factor (SF) $(mg/kg/day)^{-1}$ | Weight-of-Evidence Classification (b) | Type of Cancer | SF Source |
|---|---|---|---|---|
| Oral Route | | | | |
| Organics: | | | | |
| Benzene | 2.9E-2 | A | Leukemia | IRIS* |
| Benzo(a)pyrene (Equivalent) | 1.2E+1 | B2 | Stomach | HEAST** |
| Chlordane (total) | 1.3E+0 | B2 | Liver | IRIS |
| Dieldrin | 1.6E+1 | B2 | Liver | IRIS |
| bis(2-Ethyhexyl)phthlate | 1.4E-2 | B2 | Liver | IRIS |
| Heptachlor Expoxide | 9.1E+0 | B2 | Liver | IRIS |
| Pentachlorophenol | 1.2E-1 | B2 | Liver | HEAST |
| 2.3.7.8-TCDD (Equivalent) | 1.5E+5 | B2 | Liver & other organs | HEAST |
| Trichloroethene | 1.1E-2 | B2 | Liver | HEAST |
| Vinyl Chloride | 1.9E+0 | A | Lung | HEAST |
| Inorganics: | | | | |
| Arsenic | 1.7E+0 | A | Lung | IRIS |
| Inhalation Route (c) | | | | |
| Benzene | 2.9E-2 | A | Leukemia | HEAST |
| Trichloroethene | 1.7E-2 | B2 | Lung | HEAST |
| Vinyl Chloride | 3.0E-1 | A | Liver | HEAST |

* IRIS data obtained March 1991.
** Fourth Quarter HEAST data used (September, 1990).
(a) No toxicity criteria were available for the following chemicals of potential concern: aluminum, cobalt, lead, dibenzofuran, endosulfan sulfate, acenaphthene, phenanthrene, 2-methylnaphthalene. Criteria on carcinogenicity were not avialable for the following chemicals: antimony, barium, chromium, manganese, nickel, thallium, vanadium, and fluoranthene.
(b) See text for weight-of-evidence classification description.
(c) Inhalation toxicity criteria presented for chemicals in groundwater that may volatilize while showering.

- variation in the human population (UF = 10);
- extrapolation from animal to human studies (UF = 10);
- derivation of a chronic RfD from a subchronic NOAEL (UF = 10); and
- derivation of a chronic RfD from a chronic LOAEL (UF = 10).

An additional safety factor, referred to as the modifying factor (MF), may be applied when deriving the RfD to account for other sources of uncertainty in the study. The modifying factor is a value that ranges from 1 to 10 which is assigned based on a qualitative evaluation of the study. RfDs are developed by the intra-agency RfD Workgroup in accordance with EPA guidelines (EPA 1986b, EPA 1989e,f).

The approach discussed above can be used to evaluate the noncarcinogenic effects associated with chemicals at the Havertown PCP site with the exception of lead. Recent studies on the noncarcinogenic effects of lead suggest that developing a RfD would not be appropriate given that the effects may not have a threshold. EPA recommends using a pharmacokinetic model known as the Integrated Uptake/Biokinetic (IU/BK) model to determine blood lead levels in children (see Section 6.1.3.4 for more information concerning the IU/BK model). The model is used to predict the proportion of the population above the interim criteria of 10 ug/dl of lead in blood. Blood lead levels in children above 10 ug/dl show indications of peripheral nerve dysfunction, indexed by slowed nerve conduction velocities (NCV) based on collective neurobehavioral studies of CNS cognitive effects. These results may be indicative of a likely association between neuropsychological defects and low-level lead exposures.

RfDs and supporting toxicity data for chemicals of potential concern are summarized in Table 6-31. Toxicity profiles for the primary chemicals of concern at the Havertown PCP site (i.e., dioxin, PAHs, and PCP) are attached. For the majority of the exposure pathways evaluated in this report, dioxin, PAHs, and PCP accounted for over 95 percent of the total carcinogenic and noncarcinogenic risk.

Table 6-31

Chronic Noncarcinogenic Toxicity Criteria (RfDs)
for Chemicals of Concern at the Havertown PCP Site

| Chemical (a) | Chronic RfD (mg/kg/day) (oral route) | Condifence Level (b) | Critical Effect | RfD Source | Uncertainity (c) and Modifying Factors |
|---|---|---|---|---|---|
| Organics: | | | | | |
| Chlordane (total) | 6.0E-5 | Low | Regional Liver hypertropny in females | IRIS ** | UF = 1000 for H,A,S; MF = 1 |
| 1,2-Dichloroethene (total) | 2.0E-2 | - - - | Decreased hematocrit and nemoglogin, increased serum akaline phosphatase | HEAST * | UF = 1000 for H,A,S |
| Dieldrin | 5.0E-5 | Medium | Liver Lesions | IRIS | UF = 100 for H,A; MF = 1 |
| bis(2-Ethylhexyl)phthalate | 2.0E-2 | Medium | Increased relative liver weight | IRIS | UF = 1000 for H,A,S; MF = 1 |
| Fluoranthene | 4.0E-2 | - - - | Nephropathy, liver weight Changes, hematological cnanges | HEAST | UF = 300 for H,A,S |
| Heptachlor Epoxide | 1.3E-5 | Low | Increased liver-to-body weight ratio | IRIS | UF = 1000 for H,A,L; MF = 1 |
| Napnthalene | 4.0E-3 | - - - | Ocular and internal Lesions | HEAST | UF = 10,000 for H,A,S,L |
| Pentachlorophenol | 3.0E-2 | Medium | Liver and Kidney | IRIS | UF = 100 for H,A; MF = 1 |
| 2,3,7,8-TCDD (Equivalent) | 1.0E-9 | - - - | Reproduction | HA | UF = 1000 for H,A,L; MF = 1 |
| Inorganics: | | | | | |
| Antimony | 4.0E-4 | Low | Blood glucose, cholesterol | IRIS | UF = 1000 for H,A,L; MF = 1 |
| Arsenic | 1.0E-3 | - - - | Keratosis and hyperpigmentation | IRIS | UF = 1 |
| Barium | 7.0E-2 | Medium | Increased blood pressure | IRIS | UF = 3 for H; MF = 1 |
| Chromium (hexavalent) | 5.0E-3 | - - - | None observed | IRIS | UF = 500 for H,A,S |
| Manganese | 1.0E-1 | Medium | Central Nervous System Effects | IRIS | UF = 1; MF = 1 |
| Nickel | 2.0E-2 | Medium | Decreased body and organ weights | IRIS | UF = 300 for H,A,S; MF = 3 |
| Thallium | 7.0E-5 | - - - | Increased SGOT and serum LDH levels, alopecia | HEAST | UF = 3000 for H,A,S |
| Vanadium | 7.0E-3 | - - - | None observed | HEAST | UF = 100 for A,S |

- - - -- No data available
* HEAST data used from September, 1990
** IRIS data obtained March, 1991
(a) No toxicity criteria were available for the following chemicals of potential concern: aluminum, cobalt, lead, dibenzofu endosulfan sulfate, acenaphthene, phenanthene, and 2-methylnaphthalene. Criteria on effects other than carcinogenicity not available for benzo(a)pyrene (equivalent) for the oral route; nor for benzene, 1,2-dichloroethene (total), trichloroeth and vinyl chloride for the inhalation route.
(b) Confidence level as given by IRIS
(c) Uncertainity adjustments represent the following combined extrapolations:
H = variation in human sensitivity;
A = animal to human extrapolation;
S = extrapolation from subchronic to chronic NOAEL; and
L = extrapolation from a LOAEL to a NOAEL.

# TOXICITY PROFILES
# FOR
# THE PRIMARY CHEMICALS OF CONCERN

# TOXICITY PROFILE FOR DIOXIN (2,3,7,8-TCDD)

## General Description

Chemical Properties:

Molecular Formula: $C_{12}H_4Cl_4O_2$ (Sax 1984)

Molecular Weight: 321.96 (Sax 1984)

Half-Life: The half-life of 2,3,7,8-TCDD in water is 1-2 years and 10-12 years in soil (EPA 1984a).

Fate: Based on available data, the vertical movement of 2,3,7,8-TCDD in soil is negligible under most conditions; however, dioxin may leach from soil with low organic content (EPA 1984a).

## Carcinogenic Effects

Hazard Identification/Dose Response Assessment - Toxicological data on chlorinated dibenzo-p-dioxins (CDDs) and chlorinated dibenzofurans (CDFs) have been compiled and evaluated in several reports (EPA 1984a, 1985c, 1988d; Ontario Ministry of the Environment 1984). Of the 210 congeners of CDDs and CDFs, the compound that appears to be the most toxic is 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Experimental studies with 2,3,7,8-TCDD in animal systems have demonstrated a variety of toxic effects resulting from exposure to this compound (EPA 1985c). These effects include carcinogenesis, cancer promotion, reproductive and developmental effects, immunotoxic effects, thymic atrophy, liver damage, and changes in the skin and thyroid. Acute exposures of sensitive species of animals to 2,3,7,8-TCDD resulted in a characteristic "wasting syndrome," followed by death. Extensive experimental studies indicate there is a marked variation among species in both the array of effects caused by 2,3,7,8-TCDD and the dose levels at which these effects are elicited (EPA 1985c, Pitot et al. 1986). Limited toxicological testing of other CDDs/CDFs has shown

that several of these compounds cause similar toxicological effects, but that higher doses are generally required to cause effects of comparable magnitude to those induced by 2,3,7,8-TCDD.

In humans, the nature and extent of effects of 2,3,7,8-TCDD are less well-defined (EPA 1985c, Ontario Ministry of the Environment 1984, Pitot et al. 1986). There is a consensus that exposure of humans to 2,3,7,8-TCDD can result in a skin condition known as chloracne, an acne-like lesion which, while not life-threatening, can be disfiguring, persistent, and refractory to treatment. Several studies of human populations exposed to chemical mixtures containing 2,3,7,8-TCDD have suggested increased frequencies of certain cancers (e.g., Hardell and Sandstrom 1979, Hardell et al. 1981, Thiess et al. 1982, MDPH 1983a, Hoar et al. 1986). However, the studies are incomplete and inconsistent (U.S. EPA 1985c, Blair 1986). There is similarly inconclusive evidence for reproductive impairment in humans exposed to 2,3,7,8-TCDD (including one study conducted in Midland Co. MI: MDPH [1983b]). Other effects in humans that have been more clearly associated with exposure to 2,3,7,8-TCDD include disturbances in lipid metabolism (Moses et al. 1984, Suskind and Hertzberg 1984) and increased frequency of gastric ulcers (Bond et al. 1983, Suskind and Hertzberg 1984).

Additional information on the toxicologic effects of CDFs in humans have been observed due to two large-scale poisoning incidents in Japan and Taiwan (Kuratsune and Shapiro 1984). The exposed individuals ingested food contaminated with a mixture of CDFs, polychlorinated biphenyls (PCBs) and polychlorinated quarterphenyls (PCQs). Comparative toxicological studies indicated that CDFs were the primary toxic agents in these poisonings and that 2,3,4,7,8-PeCDF was probably the most important single compound (Masuda and Yoshimura 1984; Kunita et al. 1984, 1985; Bandiera et al. 1982; Masuda et al. 1985; Chen et al. 1985; Miyata et al. 1985). The most important toxic signs were skin eruptions similar to those of chloracne, along with skin pigmentation and eye abnormalities (Lu and Wong 1984, Urabe and Asahi 1985). Other effects observed included changes in lipid metabolism and immune function (Okumuru et al. 1974; Chang et al. 1982a,

1982b) and persistent respiratory symptoms (Nakanishi et al. 1985). Excess frequency of liver cancer and possibly lung cancer have been reported within 15 years after exposure among males (Kuratsune et al. 1987). Reproductive effects including menstrual disturbances (Kusuda 1971), skin hyperpigmentation in infants (Yamashita and Hayashi 1985, Hsu et al. 1985) and perinatal mortality (Hsu et al. 1985) have also been reported in the literature. The fact that these effects observed in humans are qualitatively similar to those reported in animals exposed to CDFs and CDDs (McNulty 1985) provides support for the use of animal data as the basis for hazard assessment for other members of these families of compounds.

The EPA has determined that the critical endpoints for purposes of assessing risk associated with exposure to CDDs/CDFs are cancer and reproductive effects, including teratogenesis as well as other non-cancerous effects. These effects will be discussed in the following sections.

Carcinogenic Effects - The EPA Health Assessment Document on CDDs summarized evidence that 2,3,7,8-TCDD is an animal carcinogen (EPA 1985c). These findings are based on laboratory results that indicate that exposure of rats and mice to 2,3,7,8-TCDD at very low doses produces tumors at several sites, but primarily in the liver (Kociba et al. 1978, NTP 1982). On the basis of these animal studies, short-term tests and structure/activity considerations, EPA concluded that 2,3,7,8-TCDD should be regarded as a "probable" human carcinogen (EPA 1985c). The agency therefore designated 2,3,7,8-TCDD as a "$B_2$" carcinogen because there is "sufficient" evidence of carcinogenicity from animal studies, but "inadequate" evidence from human epidemiological studies (EPA 1986a).

EPA has developed a Dose-Response Assessment for 2,3,7,8-TCDD based upon data from the study by Kociba et al. (1978). EPA employed the linearized multi-stage (LMS) model to estimate an upper bound for the excess lifetime cancer risk at doses below those used in animal experiments. In order to extrapolate from dose-response data in animals to predict human risk, EPA used its standard procedure of adjusting relative doses on a body surface area basis, reflective

of relative metabolic rate (EPA 1985c). Experimental animal data were used to estimate an upper bound on the cancer slope factor for 2,3,7,8-TCDD. The cancer slope factor is equivalent to the slope of the projected linear dose-response curve in the low-dose region, adjusted to apply to humans. The cancer slope factor (referred to as $q_1^*$) for 2,3,7,8-TCDD is $1.5 \times 10^5$ $(mg/kg/day)^{-1}$ (EPA 1990e). The actual slope is not likely to exceed this upper bound estimate.

In recent years, several alternative approaches to carcinogenic risk assessment for 2,3,7,8-TCDD have been presented by scientists or regulatory agencies, both in the U.S. (Miller 1983, Kimbrough et al. 1984, Portier et al. 1984, MDH 1985, MDPH 1986, Hoel 1986, Sielken 1987, Shu et al. 1987, Thorslund et al. 1987) and in other countries (Ontario 1984, FRG 1984). Most of these assessments remain unpublished and have not been peer-reviewed. In general, they differ from the EPA dose-response assessment in one or both of two respects:

- Several assessments that utilized the linearized multi-stage model incorporated different data or made different assumptions about the way in which the data should be used. Examples include the use of different sets of tumor data as the basis for extrapolation (Kimbrough et al. 1984, Portier et al. 1984), the use of tissue concentrations as measures of dose (Portier et al. 1984), the use of mg/kg body-weight scaling (Miller 1983, Kimbrough et al. 1984, MDH 1985, MDPH 1986), or the use of different ways of averaging lifetime dose (Kimbrough et al. 1984). The most important of these differences is the use of mg/kg body-weight scaling, which results in a human cancer potency factor about 5 times lower than that derived from body-surface-area scaling. Primarily for this reason, estimates of cancer potency developed by other U.S. agencies (including the Centers for Disease Control, the Food and Drug Administration, and the States of Michigan and Minnesota) have ranged from a value near to the EPA value to a value about one order of magnitude less potent (Kimbrough et al. 1984, FDA 1983, MDH 1985, MDPH 1987). Although the selection of an interspecies scaling factor is a matter for scientific judgment, the greater retention

time of 2,3,7,8-TCDD in humans than in rats provides a rationale for the selection of the more "conservative" body-surface-area scaling factor used by EPA.

- Several assessments have been based on the assumption that 2,3,7,8-TCDD acts primarily as a cancer promoter, and on the further assumption that cancer promotion is a reversible phenomenon with a threshold-type dose-response relationship. On the basis of these assumptions, "acceptable" daily intakes for 2,3,7,8-TCDD have been proposed by applying "Uncertainty Factors" to dose-levels thought to be "Lowest-Observed-Adverse-Effect-Levels" (Ontario 1984, FRG 1984, Shu et al. 1987). Although there is evidence that 2,3,7,8-TCDD is a potent promoter and has little propensity to interact with DNA in the manner of a classical cancer initiator (Pitot et al. 1986), currently available evidence on mechanisms of cancer promotion does not support the assumption that promoting activity would be reversible and have a threshold-type dose-response relationship (Upton et al. 1985; Weinstein 1984, 1987; Yamasaki and Weinstein 1985; Gallagher 1986). Goodrow et al. (1986) have reported that cancer promotion by 2,3,7,8-TCDD is associated with its binding to receptors associated with the Ah gene locus and receptors for epidermal growth factor. Other studies have suggested that binding to one or both of these receptors results in activation of certain genes (Israel and Whitlock 1984; Whitlock et al. 1984; Jones et al. 1985, 1986; Jones 1986). There is no evidence that these molecular mechanisms would necessarily be reversible and would display threshold-type dose-response relationships. Even if receptor binding is assumed to be reversible, the fact that 2,3,7,8-TCDD is more strongly retained in human tissues than in those of other animals would have to be taken into account (Hoel 1986). Finally, the promoting effects of 2,3,7,8-TCDD might augment risks resulting from prior human exposure to initiating carcinogens. At present, there are no accepted models that can be used to predict low-dose risks resulting from these effects of 2,3,7,8-TCDD. Thorslund et al.

(1987) have presented preliminary results of a model in which 2,3,7,8-TCDD is assumed to act by causing proliferation of initiated cells, but it has not been demonstrated that this approach accurately reflects the biochemical mode of action of 2,3,7,8-TCDD in cancer causation.

For the above reasons, it remains appropriate to use the dose-response assessment for 2,3,7,8-TCDD derived by EPA (1985c), based on the linearized multistage model (LMS) with body-surface-area scaling. Portier et al. (1984) have reported that available dose-response data fit a linear model if tissue concentration is used as a measure of dose. EPA recognizes, however, that use of the LMS model is controversial at the present time; dose-response assessment for carcinogenic effects of 2,3,7,8-TCDD is currently under review by the Agency, and this review may lead to revision of the cancer potency factor. Further research and mathematical modeling will help to resolve some of this uncertainty (EPA 1988d).

Ongoing work on mechanisms of action (Jones et al. 1986, Jones 1986, Goodrow et al. 1986), pharmacokinetics (Leung et al. 1987, Van den Berg and Poiger 1987), and mathematical modeling (Thorslund et al. 1987) will eventually help to resolve the controversies surrounding cancer risk estimates for 2,3,7,8-TCDD. Pending this resolution, it should be recognized that these features of the biological activity of 2,3,7,8-TCDD add substantial uncertainty to risk estimates derived from the LMS model. These estimates are intended to represent upper bounds on risk and will be reported as such. Even as upper bound, however, they could be too high (e.g., if the dose-response relationship is strongly non-linear) or too low (e.g., if CDDs/CDFs act to promote cancers initiated by other widespread environmental carcinogens).

Chronic Reproductive Effects - The chronic RfD is based on reproductive effects resulting from long-term exposure to low levels of dioxin. 2,3,7,8-TCDD has been shown to be teratogenic in all strains of mice tested. This compound produced teratogenic and fetotoxic effects in all strains of rats tested and reproductive effects in other species, such as subhuman primates (EPA 1985c).

For reproductive effects, EPA has focused on a three-generation rat feeding study (Murray et al. 1979) as the critical study for estimating the non-cancer risk posed by 2,3,7,8-TCDD. The Centers for Disease Control (CDC) have cited a reproductive study in monkeys (Allen et al. 1979) as the critical study (Kimbrough et al. 1984). EPA (1985c) also cited this study, as well as another report on the same research (Schantz et al. 1979) in support of their findings. For teratogenic effects, the critical study is a study in rats treated with 2,3,7,8-TCDD, administered daily by gavage on days 6-15 of gestation (Sparschu et al. 1971).

There has been some debate as to whether or not a dose of as little as 1 ng/kg/d (1000 pg/kg/d) of 2,3,7,8-TCDD was a NOAEL in the three-generation reproductive study in the rat (Murray et al. 1979, Nisbet and Paxton 1982, Kimbrough et al. 1984, EPA 1985c). EPA has examined this study in detail and selected a combined UF of 1000, (including subfactors of 10 because the lowest administered dose was not a NOAEL, 10 to account for possible interspecies differences in sensitivity, and 10 to account for possible intraspecies differences in sensitivity) such that an RfD of 1 pg/kg/d is derived (EPA 1987b). EPA (1985c, 1987b) also placed weight on the study by Schantz et al. (1979), which reported adverse reproductive effects in rhesus monkeys exposed to 2,3,7,8-TCDD at about 1.5 ng/kg/d, leading to a similar value for the RfD. As noted above, the CDC selected a different critical study in deriving their functional equivalent of the RfD, but the CDC scientists obtained essentially the same value as EPA, i.e., 1-2 pg/kg/day (Kimbrough et al. 1984). Thus, the RfD of 1 pg/kg/day ($1x10^{-9}$ mg/kg/day) (EPA 1984a, 1990e) for 2,3,7,8-TCDD was used in this assessment to evaluate potential noncarcinogenic effects associated with chronic exposure.

Health Advisories for 2,3,7,8-TCDD - EPA developed One-day and Ten-day Health Advisories for protection against liver effects of 100 pg/kg/day and 10 pg/kg/d, respectively (Lee 1989). These HAs will be used in this assessment to assess less than chronic exposure. In general, RfDs are based on studies involving lifetime exposure of animals and are formally defined for comparison with

lifetime average dose rates in humans (EPA 1987b). In the case of 2,3,7,8-TCDD, the RfD is based on a three-generation reproductive study in which rats were exposed for two reproductive cycles, and another study in which rhesus monkeys were exposed for only 7 months which yielded a similar LOAEL. Hence, it is appropriate to compare this RfD with dose-rates for less-than-lifetime exposure in humans.

Hazard Identification and Dose-Response Assessment for Mixtures of CDDs/CDFs, Including 2,3,7,8-TCDD - There is a limited toxicological data base for the other CDDs and CDFs, excluding 2,3,7,8-TCDD. This section summarizes the findings of limited testing of other CDDs and CDFs for carcinogenicity and teratogenicity.

A mixture of two 2,3,7,8-substituted-HxCDDs induced liver tumors in a study using rats and mice (NCI 1980). EPA (1985c) designated this mixture as a "$B_2$" carcinogen and calculated a cancer slope factor for the mixture of $3.9 \times 10^4$ $(mg/kg/day)^{-1}$. Suggestive evidence was reported for the carcinogenicity of 2,3,7,8-TCDD when it was administered to male mice at high doses (NCI 1979). 2,3,7,8-TCDF was reported to be a potent cancer promoter in a two-stage skin cancer promotion bioassay using hairless mice, although about 20 times less potent than 2,3,7,8-TCDD tested in the same study (Poland and Knutson 1982, Poland et al. 1983). 2,3,4,7,8-PeCDF and 1,2,3,4,7,8-HxCDF were reported to be potent cancer promoters in a two-stage liver cancer promotion bioassay, although the penta-substituted furan was more potent than the hexa-substituted furan (Nishizumi and Masuda 1986).

Only limited testing for teratogenic effects (and none for other reproductive effects) has been conducted for other CDDs and CDFs. 2,3,7,8-TCDF induced cleft palates and hydronephrosis in fetal mice when it was administered on days 10-13 of gestation (Weber et al. 1984, Hassoun et al. 1984, Krowke 1986). 1,2,3,7,8-PeCDD and 1,2,3,4,7,8-HxCDD also induced cleft palates in mice when they were exposed in utero (Krowke 1986). 1,2,3,7,8-PeCDF; 2,3,4,7,8-PeCDF and 1,2,3,4,7,8-HxCDF also induced cleft palates and hydronephrosis in mice exposed

in utero (Birnbaum et al. 1987a,b). All of these effects were similar to those induced by 2,3,7,8-TCDD in the same or in parallel experiments, but 2,3,7,8-TCDD was the most potent of the compounds tested in these respects.

Other toxicologic studies using bioassay systems, primarily with the liver and thymus, have demonstrated that most CDDs and CDFs produce effects similar to 2,3,7,8-TCDD, but 2,3,7,8-TCDD is the most potent congener tested (McKinney and McConnell 1982; Mason et al. 1985, 1986a,b; Safe 1986). These studies have shown structure-activity relationships within both families of compounds, with a general parallelism between relative potencies in in vivo and in vitro bioassays (Safe 1986). The results of these studies have suggested a general approach to risk assessment for these compounds which can be applied to complex mixtures of the type commonly found in the environment.

Toxicity Equivalence Factors - EPA adopted a science policy position for assessing risks of congeners and isomers of CDDs/CDFs using "toxicity equivalence factors" (TEFs) (EPA 1989g, Thomas 1987). The procedure is based on the toxicologic finding that the family of furans and dioxins has similar toxicologic signs but differ in their relative potencies. The procedure underwent internal and external EPA review, including examination by the EPA's Science Advisory Board (SAB 1986). It has been adopted by EPA as an interim procedure to be used until sufficient additional data are available to derive a more accurate procedure that can be scientifically validated. The TEF approach uses similarity in structure and activity as the basis for estimating the toxicity of any CDD/CDF mixture in terms of an equivalent amount of 2,3,7,8-TCDD. The basic method used to quantitate a mixture containing the dioxins and furans is outlined in EPA (1988c). The method assigns a TEF of one to 2,3,7,8-TCDD and lesser TEFs to the other members of the family, depending on their toxicities relative to 2,3,7,8-TCDD. Structure-activity studies have shown that 2,3,7,8-substituted congeners are more potent in a number of assays than non-2,3,7,8-substituted congeners (Poland et al. 1979; Mason et al. 1985, 1986a,b; Safe 1986) and the former are assigned much higher TEFs.

The TEF approach is used in this risk assessment to convert reported quantities of 2,3,7,8-TCDF in monitoring samples to 2,3,7,8-TCDD (Equivalent). The resulting concentrations of 2,3,7,8-TCDD (Equivalent) are then treated as if they were concentrations of 2,3,7,8-TCDD itself. The TEF procedure incorporates a number of assumptions with varying scientific basis and degree of validation; these assumptions are listed below with comments on their basis and limitations.

1. All CDD/CDF congeners have the same mechanism of action and cause the same spectrum of toxic effects; there is an extensive empirical basis for this assumption, at least for mechanisms of action and acute toxic effects (Safe 1986, EPA 1989g).

2. The relative potencies of the CDD/CDF congeners are similar for different toxic effects, so that measures of relative potency derived from in vitro or short-term in vivo tests can be used to predict relative potencies for the critical toxic effects used in risk assessment; there is a fairly extensive empirical basis for similarity in relative potencies between in vitro and short-term in vivo measures of activity (Safe 1986); only a few CDD/CDF congeners have been tested for carcinogenicity and teratogenicity, but the results of these tests are consistent with the assumption (see references cited above).

3. The effects of different CDD/CDF congeners are additive; two in vitro studies (Sawyer et al. 1983, Safe et al. 1986) and one teratogenicity study (Krowke 1986) provide very limited support for this assumption, although two other teratogenicity studies (Weber et al. 1985, Birnbaum et al. 1987b) suggested synergistic action.

4. Within each congener group, all 2,3,7,8-TCDD-substituted congeners have similar relative potencies; however, available studies actually suggest moderate variability, sometimes by an order of magnitude (Poland et al. 1979; Knutson and Poland 1981; Mason et al. 1985, 1986a,b; Safe 1986).

5. All CDD/CDF congeners with 1-3 chlorine atoms substituted at any position have negligible biological activity; available studies suggest a low level of activity, at least for 237-substituted congeners (NCI 1979, Knutson and Poland 1981, Mason et al. 1985).

Because of the limited validation available for these assumptions, the TEF procedure is recognized to yield risk estimates with a substantial degree of uncertainty; however, it is believed that the estimates of 2,3,7,8-TCDD (Equivalent) are generally reliable to within at least an order of magnitude (EPA 1989g).

Ongoing Evaluation of Toxicity of Dioxin - The potential of TCDD and related compounds to cause cancer in humans remains an issue of considerable scientific controversy. EPA originally evaluated the toxicity of dioxin in 1985 (EPA 1985c). In this Health Assessment document, EPA presented the scientific evidence for its decision to classify TCDD as a probable human carcinogen and provided an estimated upper-limit slope factor. This estimate of TCDD potency is greater than that estimated by any other government agency, foreign or domestic. Since 1985, EPA has based all of its risk-related dioxin decisions on this 1985 upper-limit estimate.

In 1988, EPA published a draft report based on a reexamination of TCDD toxicity entitled "A Cancer Risk-Specific Dose Estimate for 2,3,7,8-TCDD," which was reviewed by EPA's Science Advisory Board (SAB). SAB concluded "that at the present time the important new scientific evidence about TCDD does not compel a change in the current assessment of the carcinogenic risk of dioxin to humans," and found "no scientific basis at this time for the proposed change in [the upper limit potency factor] for the causation of cancer by TCDD." SAB also made several recommendations to EPA regarding additional efforts for improving its TCDD risk estimate which EPA is in the process of implementing.

In addition, three significant events have recently occurred that relate to

dioxin toxicity. First, a report will soon be issued that summarizes the findings on dioxin toxicity from an international conference (Banbury Conference) held in October 1990. This conference was established to review the current state of knowledge on dioxin toxicity and its implications for risk assessment. Second, the National Institute of Occupational Safety and Health (NIOSH) has recently published the results of a major retrospective cohort study of approximately 5000 chemical workers occupationally exposed to TCDD contaminated chemical production processes (Fingerhut et al. 1990). This, the largest and most comprehensive epidemiology study to date of a TCDD-exposed population, found little increase in mortality from cancers previously associated with exposure of humans to TCDD, with the exception of soft-tissue sarcoma. There was, however, a small but significant increase in mortality from all cancers combined, consistent with a carcinogenic effect of TCDD. These conclusions were limited by the small number of cases, variability in pathological diagnoses, misclassified death certificates, and occupational exposures to substances other than TCDD. In addition, a 34-year mortality follow-up study of German workers exposed accidentally to 2,3,7,8-TCDD in 1953 has reported similar results (Zober et al. 1990). This was the only other cohort study which had both substantial exposure to TCDD and a long period of latency during which mortality was examined. EPA intends to incorporate the information gathered from these sources into its ongoing dioxin toxicity reevaluation as well as other information as it becomes available.

In developing and implementing EPA's dioxin risk management program, the Agency continues to use the 1985 report as its basis for dioxin risk estimates. Because of the need to evaluate all of the new evidence on TCDD, EPA concludes that it is inappropriate to initiate a major expansion or reevaluation of its current dioxin risk management efforts at this time. EPA will carefully consider any information developed during its risk assessment or risk management activities that indicates that its program direction should be changed.

## Summary Of Toxicity Criteria

Carcinogenic Toxicity:

Oral Slope Factor: $1.5 \times 10^{5}$ mg/kg/day$^{-1}$

Weight of Evidence: B2

Noncarcinogenic Toxicity:

Oral Reference Dose: $1.0 \times 10^{-9}$ (EPA 1984)

Oral Uncertainty Factor: 1000

Critical Effect: Reproduction

# TOXICITY PROFILE FOR PENTACHLOROPHENOL (PCP)

## General Description

Chemical Properties:

Molecular Weight: 266.35

Solubility: in water at 20°C = 14 mg/l (Verschueren, 1983)

Half-Lives in: Air: Unknown (EPA, 1984a)
Water: 14 days (Boyle et al., 1980)
Soil: 48 days (Rao and Davidson, 1982)

Fate: Mobility in soil is uncertain but is reported to be dependent on soil pH and organic matter content. PCP is likely to be sorbed strongly to organic-rich acidic soils, and leached from neutral soils having low organic matter content (EPA 1985d).

Absorption: PCP is rapidly absorbed from the GI tract. Reported average half-life for absorption of PCP in human volunteers is 1.3 ± 0.4 hrs after administration of 0.1 mg/kg bw (Braun et al., 1978). Casarett et al. (1969) demonstrated that PCP is also rapidly absorbed by inhalation. Two male workers were exposed to PCP for 45 minutes in an enclosed area of a wood processing plant. Mean urinary concentrations of 230 ng/l and 432 ng/l PCP were recorded and the absorption of PCP was estimated to be 88 and 76%, respectively, of the inhaled dose.

## Carcinogenic Effects

The NTP (National Toxicology Program) performed two-year dietary studies on carcinogenicity of PCP in mice using technical grade and Dowicide EC-7 (NTP, 1989). Results showed tumor development in the liver, adrenal, and circulatory

systems. EPA has recently released a slope factor for PCP of 0.12 mg/kg/day$^{-1}$ (EPA 1990e, 1991e). No human studies demonstrating carcinogenic activity for PCP were found in the available literature. PCP has been classified as a probable human carcinogen "B2" (EPA 1990e, 1991e).

Noncarcinogenic Effects

No human studies were found in the available literature. Studies dosing rats and hamsters with PCP showed dose-related fetal toxicity (Larsen et al., 1975; Schwetz and Gehring, 1973; Schwetz et al., 1974a,b; Schwetz et al., 1978; Hinkle, 1973). Only one chronic study (Schwetz et al. 1978) was found in the available literature (EPA 1991e). Twenty-five rats were administered one of three doses (3 mg/kg/day, 10 mg/kg/day, or 30 mg/kg/day) with the following results:

| | |
|---|---|
| 3 mg/kg/day: | No apparent adverse effects noted; |
| 10 mg/kg/day: | Pigmentation of the liver and kidneys in females; and |
| 30 mg/kg/day: | Reduced body weight gain and increased specific gravity of the urine in females; and pigmentation of the liver and kidneys in both females and males. |

Based on this study a NOAEL of 3 mg/kg/day was established. A RfD of 0.03 mg/kg/day was derived using the NOAEL and an uncertainty factor of 100 (EPA 1991e). The critical effect noted from the study was pigmentation of the liver and kidneys (EPA 1991e).

A number of studies investigating the teratogenicity of orally administered PCP in rodents are available in the literature. Although these studies (Larsen et al. 1975; Schwetz and Gehring 1973, Schwetz et al. 1978, Hinkle, 1973) did not reveal teratogenic effects, feto-maternal toxicity was seen at 30 mg/kg/day (Schwetz and Gehring, 1973). Since PCP apparently does not cross the placental

barrier, the observed fetotoxicity may be a reflection of maternal toxicity (Larsen et al. 1975, EPA 1991e).

Summary Of Toxicity Criteria

Carcinogenic Toxicity:

Oral Slope Factor: 0.12 mg/kg/day$^{-1}$ (EPA 1991e)
Weight of Evidence: B2

Noncarcinogenic Toxicity:

Oral Reference Dose: 0.3 mg/kg/day (EPA 1991e)
Oral Uncertainty Factor: 100 (EPA 1991e)
Target Organ: liver and kidneys
Critical Effect: pigmentation of the liver and kidneys

# TOXICITY PROFILE FOR PAHs

## General Description

Chemical Properties: Polycyclic Aromatic Hydrocarbons (PAHs) are a class of compounds which are formed during the incomplete combustion or pyrolysis of organic materials containing carbon and hydrogen. PAHs generally have low water solubility, very low vapor pressures, and high organic carbon partitioning coefficients.

Degradation: The removal of PAHs from the atmosphere can occur through photochemical reactions, chemical reactions (principally with OH radicals, ozone and NO2) and physical removal mechanisms (wet and dry deposition) (Atkinson 1984, HAS 1983, Mabey et al. 1981).

Fate: The primary removal mechanism for benzo(a)anthracene and benzo(a)pyrene from the atmosphere is likely to be ozonolysis reactions. The three likely mechanisms that may be responsible for the removal of PAHs from aquatic media are volatilization, photochemical reactions and microbial degradation. With the exception of naphthalene and other PAHs that have relatively high vapor pressures, volatilization is not likely to be a significant removal mechanism. In the case of naphthalene, both volatilization and adsorption may be quite competitive, with the dominant process being dictated by the aquatic conditions. High stream and wind velocities could enhance volatilization, while high organic carbon content could facilitate sedimentation and the subsequent microbial degradation of particle-sorbed naphthalene (EPA 1984b).

The predominant mechanism that is likely to dictate the fate of most PAHs in aquatic media is sorption onto particulate matter and subsequent sedimentation and microbial degradation (EPA 1984b).

The predominant mechanism for the removal of PAHs from soils is likely to be microbial degradation. Considering the soil sorption coefficient (Kenaga and Goring 1980) and water solubilities, these compounds are not expected to have high mobility in soils. Therefore, significant leaching of these compounds into groundwater is not expected, particularly from soils with higher organic carbon content (EPA 1984b).

Of the PAHs detected at the Havertown PCP site, carcinogenic PAHs (i.e., benzo(a)pyrene [Equivalent]) and naphthalene contributed significantly to carcinogenic and noncarcinogenic risk, respectively. Studies of the carcinogenicity of PAHs and the noncarcinogenic risk associated with naphthalene will be discussed below.

Carcinogenic Effects

International Agency for Research on Cancer (IARC) has judged the following specific PAHs to be probable human carcinogens, because there is sufficient animal evidence and/or limited human evidence. The EPA (1984b) has placed the following chemicals in Group B1 (Probable Human Carcinogens: Limited evidence of carcinogenicity in humans from epidemiological studies) or Group B2 (Probable Human Carcinogens: Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans), depending on the quality of the evidence:

1. benzo(a)anthracene
2. benzo(b)fluoranthene
3. benzo(j)fluoranthene
4. benzo(k)fluoranthene
5. benzo(a)pyrene
6. dibenz(a,h)acridine
7. dibenz(a,j)acridine
8. dibenzo(a,h)anthracene
9. dibenzo(c,g)carbazo

10. dibenzo(a,e)pyrene
11. dibenzo(a,h)pyrene
12. dibenzo(a,i)pyrene
13. dibenzo(a,l)pyrene
14. indeno(1,2,3-c,d)pyrene

Also, the following compounds have limited animal evidence for carcinogenicity; however, the evidence according to IARC is inadequate for making a definitive statement about the human carcinogenic potential. The following compounds have been placed in Group C, Possible Human Carcinogens:

1. anthracene
2. benzo(c)acridine
3. carbazole
4. chrysene
5. cyclopenta(c,d)pyrene
6. dibenzo(a,c)anthracene
7. dibenzo(a,j)anthracene
8. dibenzo(a,e)fluoranthene
9. 2- and 3-methylfluoranthenes

Carcinogenic risk factors for PAHs are summed using the toxicity equivalence factors as substituted for benzo(a)pyrene as discussed in Section 6.1.2.

In animals, the carcinogenic properties of certain PAH compounds have been studied in animals for more than 50 years. The predominance of testing has been done with oral, inhalation exposures, mouse skin assays, implantations and subcutaneous injections. Benzo(a)pyrene administered orally in the diet to mice resulted in increased incidence of papillomas and carcinomas (stomach tumors: Neal and Rigdon (1967), as well as, lung adenoma and leukemia (Rigdon and Neal 1966, 1969). Incidence of lung adenomas and liver hepatomas was elevated in animals given benzo(a)pyrene by gavage (Klein 1963). An oral slope factor of

11.5 per mg/kg/day was derived by EPA (1991f).

Noncarcinogenic Effects

Of 7 pregnant benzo(a)pyrene-treated rats, only 1 dam carried viable fetuses to term, delivering 4 pups on the 23rd day of pregnancy. Two of the 4 pups were stillborn, one of which was grossly malformed; another pup died of starvation 3 days after birth, since the dam did not show any signs of lactation. At autopsy, 4 dead fetuses were found in the right uterine horn of a second dam (Rigdon and Rennels 1964). In another teratogenicity and reproduction study in mice, Rigdon and Neal (1965) administered diets containing benzo(a)pyrene and found no apparent reproductive teratogenic or fetotoxic effects in lab animals. Mackenzie and Angevine (1981) observed a specific reduction of gonadal weight, reduced fertility and reproductive capacity among offspring and almost complete sterility of offspring in the high dose group only of mice fed benzo(a)pyrene orally during pregnancy. Sufficient information to derive a RfD for benzo(a)pyrene were not available.

HEAST (EPA 1990e) reported a chronic RfD for naphthalene of 0.004 mg/kg/day based on a chronic rat study. Rats were administered 50 mg/kg/day of naphthalene by gavage for 5 days per week for 13 weeks. The critical effect observed in the study was a decrease in body weight. An uncertainty factor of 10,000 was applied to the dose level to derive the RfD.

Summary Of Toxicity Criteria

Carcinogenic Toxicity:

Oral Slope Factor: 11.5 mg/kg/day$^{-1}$ for benzo(a)pyrene (Equivalent) (EPA 1990e, 1991f)

Weight of Evidence: B2

Noncarcinogenic Toxicity:

Oral Reference Dose: $4 \times 10^{-3}$ mg/kg/day for naphthalene (EPA 1990e)

Oral Uncertainty Factor: 10,000

Target Organ:

Critical Effect: weight loss

### 6.1.5 Human Health Risk Assessment

The final step in the baseline risk assessment process is risk characterization. In this section, toxicity criteria identified in Section 6.1.4 are combined with exposure estimates presented in Section 6.1.3 to quantify potential noncarcinogenic and carcinogenic effects associated with chemicals of potential concern from the Havertown PCP site. Section 6.1.5.1 presents an overview of the methods for quantifying potential carcinogenic and noncarcinogenic risks. Potential risks associated with exposure pathways evaluated under current and future land-use of the Havertown PCP site are discussed in Section 6.1.5.2 and Section 6.1.5.3, respectively.

#### 6.1.5.1 Methods for Estimating Carcinogenic and Noncarcinogenic Risks

Potential carcinogenic risks are expressed as an increased probability of developing cancer over a lifetime (i.e., excess individual lifetime cancer risk) (EPA 1989a). For example, a $10^{-6}$ increased cancer risk can be interpreted as an increased risk of 1 in 1,000,000 for developing cancer over a lifetime if an individual is exposed as defined by the pathways presented in this report. A $10^{-6}$ increased cancer risk is the point of departure established in the NCP (EPA 1990a). In addition, the NCP (EPA 1990a) states that "for known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between $10^{-4}$ and $10^{-6}$."

Carcinogenic risks for chemicals of potential concern are quantified using the equation below:

$$Cancer\ Risk_i = CDI_i * SF_i$$

where:

Cancer Risk$_i$ = The potential carcinogenic risks associated with exposure to chemical$_i$ (unitless);

$CDI_i$ = Chronic daily intake for chemical$_i$ (mg/kg/day); and
$SF_i$ = Slope Factor for chemical$_i$ $(mg/kg/day)^{-1}$.

If the carcinogenic risk exceeds $10^{-2}$, then EPA (1989a) guidance recommends using the following equation to estimate carcinogenic risk:

$$Cancer\ Risk_i = 1 - e^{(-CDI_i * SF_i)}$$

where:

Cancer Risk$_i$ = Increased carcinogenic risk associated with exposure to chemical$_i$ (unitless);
$CDI_i$ = Chronic daily intake for chemical$_i$ (mg/kg/day); and
$SF_i$ = Slope Factor for chemical$_i$ $(mg/kg/day)^{-1}$.

Chemical-specific cancer risks are summed in accordance with EPA (1989a, 1986a,b) guidance in order to quantify the combined cancer risk associated with exposure to a chemical mixture. The slope factor is the 95th UCL on the linear slope that describes the cancer potency of the chemical of potential concern. Using the 95th UCL on the linear slope is a conservative approach adopted by the EPA in order that the true risks will not be underestimated.

Noncarcinogenic effects are not quantified as a probability of exhibiting a particular effect. Rather, noncarcinogenic effects are evaluated by comparing the estimated dose (i.e., CDI) with a reference dose (RfD). The hazard quotient is used to quantify the potential for an adverse noncarcinogenic effect to occur and is calculated using the following equation:

$$HQ_i = \frac{CDI_i}{RfD_i}$$

where:

$HQ_i$ = Hazard quotient for $chemical_i$ (unitless);
$CDI_i$ = Chronic Daily Intake for $chemical_i$ (mg/kg/day); and
$RfD_i$ = Reference Dose for $chemical_i$ (mg/kg/day).

If the hazard quotient exceeds unity (i.e., 1), then an adverse health effect may occur. The higher the hazard quotient the more likely that an adverse noncarcinogenic effect will occur as a result of exposure to the chemical of potential concern. If the estimated hazard quotient is less than unity, then an adverse noncarcinogenic effect is unlikely to occur.

EPA (1989a, 1986b) recommends summing chemical-specific hazard quotients to evaluate the combined noncarcinogenic risk from exposure to a chemical mixture. The sum of the chemical-specific hazard quotients is called the hazard index. Using this approach assumes that chemical-specific noncarcinogenic risks are additive. Limited data are available for actually quantifying the potential synergistic and/or antagonistic relationships between chemicals in a chemical mixture. In addition, it is assumed that the target organs and toxicological mechanisms that may result in the effect are the same for all chemicals evaluated in the chemical mixture. If the latter assumption is not valid and the hazard index exceeds unity, then hazard indices should be calculated by target organ and mechanism, as recommended by EPA (1989a) guidance.

The following sections present carcinogenic risks and hazard quotients for chemicals of potential concern for the RME case for pathways under current land-use and future land-use conditions.

6.1.5.2 Potential Risks Under Current Land-Use Conditions

Direct Contact with Surface Water by Children Playing in Naylors Run - Potential carcinogenic risks to children playing in Naylors Run due to dermal absorption of chemicals of potential concern in surface water are presented in Table 6-32. Five probable human carcinogens (Group B2) were detected in surface water samples

Table 6-32

Potential Carcinogenic Risk Associated with
Direct Contact with Surface Water from Naylors
Run by Children for the RME Case

| Chemical | RME Chronic Daily Intake (mg/kg/day) | Slope Factor $(mg/kg/day)^{-1}$ | Weight-of-Evidence | Potential Cancer Risk |
|---|---|---|---|---|
| Organics: | | | | |
| Dieldrin | 1.3E-9 | 1.6E+1 | B2 | 2.1E-8 |
| Heptachlor Epoxide | 3.4E-9 | 9.1E+0 | B2 | 3.1E-8 |
| Benzo(a)pyrene (Equivalent) | 1.3E-9 | 1.2E+1 | B2 | 1.6E-8 |
| Pentachlorophenol | 5.2E-6 | 1.2E-1 | B2 | 6.2E-7 |
| 2,3,7,8-TCDD (Equivalent) | 1.2E-12 | 1.5E+5 | B2 | 1.8E-7 |
| | | | Total Carcinogenic Risk | 9.0E-7 |

from Naylors Run including dieldrin, heptachlor epoxide, benzo(a)pyrene (Equivalent), PCP, and 2,3,7,8-TCDD (Equivalent). The total carcinogenic risk associated with dermal absorption of these chemicals was $9x10^{-7}$. The potential carcinogenic risk associated with direct contact with surface water was below the point of departure established in the NCP (EPA 1990a). The majority of the carcinogenic risk was associated with dermal absorption of PCP and 2,3,7,8-TCDD (Equivalent). The maximum detected concentrations for these chemicals were found in the catch basin which is currently fenced (in this report data from the catch basin were included when estimating exposure). Therefore, potential exposure to these chemicals of concern may be overestimated given that the fence prevents access to areas with higher surface water contamination. Overall, surface water in Naylors Run does not appear to present an appreciable carcinogenic risk to children who may play in this stream given the low risks estimated for this pathway and the conservative assumptions used to assess exposure (e.g., high frequency of exposure, playing exclusively in the most contaminated area at the site, etc.).

Potential noncarcinogenic risks to children playing in Naylors Run due to dermal absorption of chemicals of potential concern in surface water are presented in Table 6-33. All of the chemical-specific hazard quotients were nearly 3 orders of magnitude below unity (1). In addition, the hazard index was nearly 2 orders of magnitude below unity. Therefore, it is highly unlikely that noncarcinogenic effects would occur in children from dermal absorption of chemicals of concern in surface water during playing activities.

Direct Contact with Sediments by Children Playing in Naylors Run - Potential carcinogenic risks to children playing in Naylors Run due to dermal absorption and incidental ingestion of chemicals of potential concern in sediments are presented in Table 6-34. Organic probable human carcinogens of concern (Group B2) were detected in sediment samples from Naylors Run including

Table 6-33

Potential Noncarcinogenic Risks Associated with Direct Contact with Surface Water by Children Playing in Naylors Run for the RME Case

| Chemical (a) | RME Chronic Daily Intake (m/d/kg/day) | RfD (mg/kg/day) | RfD Uncertainty Factor | Hazard Quotient |
|---|---|---|---|---|
| Organics: | | | | |
| Dieldrin | 9.0E-9 | 5.0E-5 | 100 | 1.8E-4 |
| Heptachlor Epoxide | 2.4E-8 | 1.3E-5 | 1000 | 1.8E-3 |
| Pentachlorophenol | 3.6E-5 | 3.0E-2 | 100 | 1.2E-3 |
| 2,3,7,8-TCDD | 8.7E-12 | 1.0E-9 | 1000 | 8.7E-3 |
| Inorganics: | | | | |
| Manganese | 3.0E-4 | 1.0E-1 | 1 | 3.0E-3 |
| Thallium | 9.9E-8 | 7.0E-5 | 3000 | 1.4E-3 |
| | | | Total Hazard Index | 2.0E-2 |

(a) Noncarcinogenic toxicity criteria were not available for cobalt and lead, therefore hazard quotients were not estimated for these elements.

Table 6-34

Potential Carcinogenic Risk Associated with Direct Contact with Sediments for Children Playing in Naylors Run for the RME Case

| Chemical (a) | RME CDI for Incidental Ingestion (mg/kg/day) | RME CDI for Dermal Absorption (mg/kg/day) | Slope Factor $(mg/kg/day)^{-1}$ | Weight-of-Evidence | Potential Cancer Risk for Ingestion | Potential Cancer Risk for Dermal Absorption |
|---|---|---|---|---|---|---|
| Organics: | | | | | | |
| Benzo(a)pyrene (Equivalent) | 3.9E-6 | 3.9E-6 | 1.2E+1 | B2 | 4.7E-5 | 4.7E-5 |
| Chlordane (total) | 3.2E-8 | 3.2E-8 | 1.3E+0 | B2 | 4.2E-8 | 4.2E-8 |
| Pentachlorophenol | 4.2E-7 | 4.2E-7 | 1.2E-1 | B2 | 5.0E-8 | 5.0E-8 |
| 2,3,7,8-TCDD (Equivalent) | 1.7E-11 | 1.7E-11 | 1.5E+5 | B2 | 2.6E-6 | 2.6E-6 |
| Inorganics (a): | | | | | | |
| Arsenic | 8.3E-6 | - - - | 1.7E+0 | A | 1.4E-5 | - - - |
| | | Total Carcinogenic Risk by Route: | | | 6.4E-5 | 5.0E-5 |
| | | Total Carcinogenic Risk for Sediment: | | | 1E-4 | |

(a) Inorganics are not considered to be dermally absorbed and are not used in estimating risk for this pathway.

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benzo(a)pyrene (Equivalent), chlordane, PCP, and 2,3,7,8-TCDD (Equivalent). In addition, arsenic which is a known human carcinogen was detected in sediment samples from Naylors Run. As presented in Table 6-34, the exposure and risk associated with dermal absorption and incidental ingestion of the organic chemicals of concern were the same. The total carcinogenic risk associated with dermal absorption of the chemicals of concern in sediment was $6x10^{-5}$. The majority of the carcinogenic risk was associated with benzo(a)pyrene (Equivalent) and arsenic. The total carcinogenic risk associated with incidental ingestion of the chemicals of concern in sediment was $5x10^{-5}$. The majority of the carcinogenic risk was associated with benzo(a)pyrene (Equivalent) (the dermal absorption of arsenic was assumed to be negligible). The highest detected concentrations of benzo(a)pyrene (Equivalent) and arsenic were found upstream of the catch basin in samples collected in Naylors Run along Eagle Road. Therefore, these locations are more accessible than the catch basin locations. The total potential carcinogenic risk associated with contact with sediment was $1x10^{-4}$ which is above the NCP point of departure (i.e., $10^{-6}$) and on the upper-bound of the NCP acceptable risk range (i.e., $10^{-4}$) (EPA 1990a). Therefore, children who engage in the activities outlined for this pathway, as discussed in Section 6.1.3, in locations upstream of the catch basin may experience an increased cancer risk level of $1x10^{-4}$. It should be noted, however, that conservative methods were used to estimate exposure to children playing in Naylors Run (e.g., high frequency of exposure, playing exclusively in the most contaminated area at the site, etc.).

Potential noncarcinogenic risks to children playing in Naylors Run due to dermal absorption and incidental ingestion of chemicals of potential concern in sediment are presented in Table 6-35. All of the chemical-specific hazard quotients were below unity (1). 2,3,7,8-TCDD (Equivalents) and chromium were the only chemicals to have a hazard quotient above 0.1. The highest detected concentrations of 2,3,7,8-TCDD (Equivalent) were found at the catch basin and directly outside of the catch basin. The highest detected concentrations of chromium were found upstream of the catch basin. To be conservative, it was assumed that chromium

Table 6-35

Potential Noncarcinogenic Risks Associated with Direct Contact
with Sediments for Children Playing in Naylors Run
for the RME Case

| Chemical (a) | RME CDI for Incidental Ingestion (mg/kg/day) | RME CDI for Dermal Absorption (mg/kg/day) | RfD (mg/kg/day) | RfD Uncertainty Factor | Hazard Quotient for Ingestion | Hazard Quotient for Dermal Absorption |
|---|---|---|---|---|---|---|
| Organics: | | | | | | |
| Chlordane (total) | 2.2E-7 | 2.3E-7 | 6.0E-5 | 1000 | 3.7E-3 | 3.8E-3 |
| Fluoranthene | 2.0E-5 | 2.1E-5 | 4.0E-3 | 300 | 5.0E-3 | 5.3E-3 |
| Pentachlorophenol | 2.9E-6 | 3.0E-6 | 3.0E-2 | 100 | 9.7E-5 | 1.0E-4 |
| 2,3,7,8-TCDD (Equivalent) | 1.2E-10 | 1.2E-10 | 1.0E-9 | 1000 | 1.2E-1 | 1.2E-1 |
| Inorganics (b): | | | | | | |
| Antimony | 2.1E-5 | - - - | 4.0E-4 | 1000 | 9.5E-2 | - - - |
| Arsenic | 5.6E-5 | - - - | 1.0E-3 | 1 | 5.6E-2 | - - - |
| Barium | 6.2E-4 | - - - | 5.0E-2 | 3 | 3.2E-2 | - - - |
| Chromium | 8.0E-4 | - - - | 5.0E-3 | 500 | 2.8E-1 | - - - |
| Manganese | 7.1E-3 | - - - | 1.0E-1 | 1 | 7.1E-2 | - - - |
| Nickel | 5.0E-5 | - - - | 2.0E-2 | 300 | 2.5E-3 | - - - |
| Thallium | 1.5E-6 | - - - | 7.0E-5 | 3000 | 2.1E-2 | - - - |
| Vanadium | 1.8E-4 | - - - | 7.0E-3 | 100 | 2.6E-2 | - - - |
| | | | Total Hazard Index by Route: | | <1 (7.1E-1) | <1 (1.3E-1) |
| | | | Total Hazard Index for Sediment: | | <1 (8.4E-1) | |

(a) Noncarcinogenic toxicity criteria were not available for benzo(a)pyrene (equivalent), dibenzofuran, endosulfan sulfate, acenaphthene, and phenanthrene; therefore, hazard quotients were not estimated for these chemicals.

(b) The dermal absorption of inorganics is negligible; therefore, exposure and risk were not estimated for this route.

was in the hexavalent state. The hazard indices for dermal absorption and incidental ingestion were 0.1 and 0.7, respectively. The total hazard index for exposure to sediment was 0.8. Therefore, noncarcinogenic effects may not occur in children from dermal absorption and incidental ingestion of chemicals of concern in sediment during playing activities.

As discussed in Section 6.1.3, the potential noncarcinogenic risk associated with exposure to lead in sediments was evaluated using a pharmacokinetic approach. The exposure point concentration for lead in sediments was used in the soil ingestion module to estimate increased blood-lead levels due to exposure to sediments. Lead was not a chemical of concern in any other media; therefore, default parameter values were used to estimate exposure to lead from other media (i.e., drinking water, air, etc.). Figure 6-1 presents a probability density function versus blood lead concentrations for children between the ages of 0 and 7 years. The cut-off value of 10 µg/dl (vertical line) is the interim criterion for evaluating the potential risk to children from elevated blood lead levels (EPA 1991c). Children with blood lead levels in excess of 10 µg/dl may experience adverse effects associated with neurological development (see Section 6.1.4.2 for further discussion). As shown in Figure 6-1, there is a 9 percent chance that a child engaged in the activity outlined for this pathway would have a blood-lead level above 10 µg/dl. The highest detected concentrations of lead in sediments were found upstream of the catch basin. The maximum detected concentration of lead was 694 mg/kg. This level slightly exceeds the interim soil lead cleanup level at Superfund sites of 500 mg/kg which is considered protective for direct contact in residential settings (EPA 1989h).

Ingestion of Fish from Cobbs Creek - Potential carcinogenic risks to recreational fisherman that ingest fish caught from Cobbs Creek are presented in Table 6-36. Four probable human carcinogens of concern (Group B2) were detected in white sucker tissue sampled from Cobbs Creek including chlordane, dieldrin, heptachlor epoxide, and 2,3,7,8-TCDD (Equivalent). Fish tissue samples which were obtained from the National Bioaccumulation Study (NBS) were not analyzed for PCP or PAHs.

FIGURE 6-1

Distribution of Possible Blood Lead Concentrations
in Children from Contact with Sediments from Naylors Run

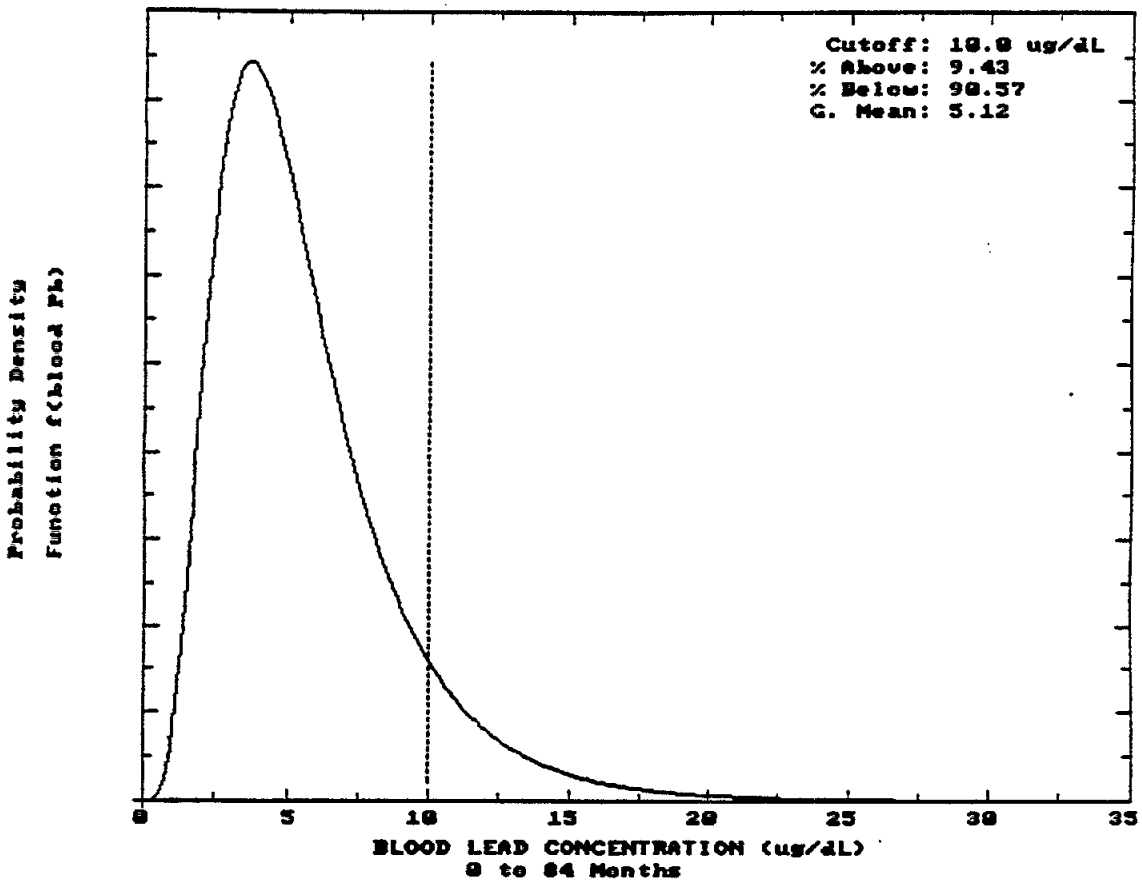


Table 6-36

Potential Carcinogenic Risks Associated with Ingestion
of Fish from Cobbs Creek Downstream
of the Havertown PCP Site (a)

| Chemical (a) | RME Chronic Daily Intake (mg/kg/day) | Slope Factor $(mg/kg/day)^{-1}$ | Weight of-Evidence | Potential Cancer Risk |
|---|---|---|---|---|
| Chlordane (total) | 6.1E-5 | 1.3E+0 | B2 | 7.9E-5 |
| Dieldrin | 1.2E-4 | 1.6E+1 | B2 | 1.9E-3 |
| Heptachlor Epoxide | 9.5E-6 | 9.1E+0 | B2 | 8.6E-5 |
| 2,3,7,8-TCDD (Equivalent) | 1.8E-9 | 1.5E+5 | B2 | 2.7E-4 |
| | Total Carcinogenic Risk | | | 2E-3 |

(a) Exposure and risk associated with ingestion of white suckers which are bottom feeders. Exposure associated with ingestion of sport fish such as bass may be much lower given their foraging behavior. The risks are only for chemicals that may be attributed to releases from the site. Risks from exposure to other chemicals in fish tissue (e.g., PCBs) which were not detected in surface water or sediments were not included in this assessment.

Other probable carcinogens were detected in fish tissue including PCBs; however, they were not included in the assessment of risk since these were not detected in surface water or sediments in Naylors Run. The total carcinogenic risk associated with ingestion of fish tissue for the RME case was $2x10^{-3}$. The majority of the carcinogenic risk was associated with dieldrin. Dieldrin was detected in Naylors Run surface water, but not in any other media. It is uncertain whether dieldrin or other chemicals of concern present in fish tissue is associated with chemical releases from the Havertown PCP site or other sources (e.g., surface water run-off, landfill). The total potential carcinogenic risk associated with ingestion of fish tissue is above the NCP point of departure (i.e., $10^{-6}$) and the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). It should be noted, however, that it is unlikely that recreational fisherman would ingest large quantities of a bottom feeding fish such as the white sucker. Exposure and risk associated with ingestion of game fish such as bass may be much lower since game fish may have much lower levels of chemicals of concern given their foraging behavior (i.e., less contact with contaminated sediments). This generalization was made based on a comparison between chemical concentrations in bottom feeding fish relative to game fish presented in the NBS. It cannot be ruled out, however, in certain well developed ecological communities game fish may have higher levels due to food chain accumulation. Fish tissue samples from game fish, however, were not available for inclusion in this risk assessment. It should be noted, however, that whole body analysis was used to monitor white suckers tissue. This may underestimate exposure since chemicals tend to partition more in fat tissue portions of the filet.

Potential noncarcinogenic risks to recreational fisherman that ingest fish caught from Cobbs Creek are presented in Table 6-37. All of the chemical-specific hazard quotients exceeded unity (1). The hazard index for ingestion of fish was 14. Therefore, ingestion of large quantities of white sucker from Cobbs Creek may result in a noncarcinogenic effect. Given the conservative assumptions discussed above, it is unlikely that recreational fisherman are actually being

Table 6-37

Potential Noncarcinogenic Risks Associated with
Ingestion of Fish from Cobbs Creek Downstream
of the Havertown PCP Site (a)

| Chemical (a) | RME Chronic Daily Intake (mg/kg/day) | Reference Dose (mg/kg/day) | Weight of-Evidence | Hazard Quotient |
|---|---|---|---|---|
| Chlordane (total) | 1.4E-4 | 6.0E-5 | 1000 | 2.3 |
| Dieldrin | 2.7E-4 | 5.0E-5 | 100 | 5.4 |
| Heptachlor Epoxide | 2.2E-5 | 1.3E-5 | 1000 | 1.7 |
| 2,3,7,8-TCDD (Equivalent) | 4.2E-9 | 1.0E-9 | 1000 | 4.2 |
| | Total Noncarcinogenic Risk | | | 13.6 |

(a) Exposure and risk associated with ingestion of white suckers which are bottom feeders. Exposure associated with ingestion of sport fish such as bass may be much lower given their foraging behavior. The risks are only for chemicals that may be attributed to releases from the site. Risks from exposure to other chemicals in fish tissue (e.g., PCBs) which were not detected in surface water or sediments were not included in this assessment.

exposed at such levels.

Indirect Exposure to Nursing Infants from Maternal Exposure to Fish - Nursing infants may be indirectly exposed to dioxin and furans in fish tissue via lactational transfer assuming that the mother is directly exposed to dioxin and furans in fish tissue as outlined in the pathway above. Potential exposure to nursing infants was estimated using a pharmacokinetic model that relates exposure of the mother from ingestion of fish to the exposure of the nursing infant via lactational transfer. The potential increased carcinogenic risk to nursing infants via indirect exposure is presented in Table 6-38. The increased carcinogenic risk associated with 2,3,7,8-TCDD (Equivalent) exposure for nursing infants (i.e., no additional exposure later in life) was estimated to be $1x10^{-4}$. The increased risk is above the NCP point of departure (i.e., $10^{-6}$) and is equal to the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). It should be noted, however, that exposure to nursing infants is based on the exposure to the mother which was derived using several conservative assumptions (e.g., ingesting 42 grams of bottom feeding fish per day).

Potential noncarcinogenic risk to nursing infants via indirect exposure of 2,3,7,8-TCDD (Equivalent) is presented in Table 6-39. The hazard quotient for chronic exposure (i.e., 2 year lactational exposure) exceeded unity by an order of magnitude. In addition, exposure to nursing infants exceeded the 10 day health advisory for potential liver impacts. Although, exposure to nursing infants exceeded a 10-day health advisory, it is still assumed that the exposure duration for the mother from ingestion of fish is chronic (which would result in significant bioaccumulation of dioxin in the mother prior to lactation).

Multimedia Assessment of Risk Under Current Land-Use Conditions - Potential carcinogenic risk and noncarcinogenic risk from exposure to all current land-use exposure pathways quantitatively evaluated in the risk assessment are presented in Table 6-40. The total carcinogenic risk was $2x10^{-3}$, while the hazard quotient

Table 6-38

Potential Increased Carcinogenic Risk
from Nursing Exposure to 2,3,7,8-TCDD (Equivalent)

| Maternal Exposure Pathway (a) | Nursing Infant RME CDI (mg/kg/day) | Slope Factor $(mg/kg/day)^{-1}$ | Weight-of-Evidence | Potential Increased Cancer Risk (b) |
|---|---|---|---|---|
| Current Land-Use: | | | | |
| Ingestion of Fish | 8.4E-10 | 1.5E+5 | B2 | 1.3E-4 |
| Future Land-Use: | | | | |
| Ingestion of Groundwater | 1.0E-6 | 1.5E+5 | B2 | 1.4E-1 |

(a) Pathway by which mother is exposed.
(b) Potential cancer risk to infant associated with nursing exposure only. Subsequent exposure to dioxin later in life is assumed to be zero.

Table 6-39

Potential Noncarcinogenic Risks Associated with
Nursing Infant Exposure to 2,3,7,8-TCDD (Equivalent)

| Maternal Exposure Pathway (a) | Nursing Infant RME CDI (mg/kg/day) | RfD (mg/kg/day) | RfD Uncertainty Factor | Hazard Quotient |
|---|---|---|---|---|
| Current Land-Use: | | | | |
| Ingestion of Fish | 3.3E-8 | 1.0E-9 (chronic) | 1000 | 33 |
| | | 1.0E-8 (10 day HA)(b) | 100 | 3.3 |
| | | 1.0E-7 (1 day HA)(b) | 10 | 0.3 |
| Future Land-Use: | | | | |
| Ingestion of Groundwater | 3.9E-5 | 1.0E-9 (chronic) | 1000 | 39,000 |
| | | 1.0E-8 (10 day HA)(b) | 100 | 3,900 |
| | | 1.0E-7 (1 day HA)(b) | 10 | 390 |

(a) Pathway by which mother is exposed.

(b) 10 day HA - 10 day Health Advisory for adverse liver effects (see toxicity profile)
1 day HA - 1 day Health Advisory for adverse liver effects (see toxicity profile)

Table 6-40

Potential Risks from Multiple Exposure Pathways
under Current Land-Use Conditions

| Pathway | Potential Carcinogenic Risk for the RME Case | Hazard Index for RME Case |
|---|---|---|
| Children Playing in Naylors Run: | | |
| Ingestion of Sediments | 6E-5 | 6E-1 |
| Dermal absorption from sediments | 5E-5 | 1E-1 |
| Dermal absorption from surface water | 9E-7 | 2E-2 |
| Subtotal for Pathway: | 1E-4 | 0.7 |
| Fishing in Cobbs Creek | 2E-3 | 14 |
| Nursing Infant Exposure (a) | 1E-4 | 33 |
| Total for all Routes (b): | 2E-3 | 5E+1 |

(a) Assumes that the mother ingests fish caught from Cobbs Creek according to the RME scenario outlined in this report.

(b) It should be noted that these risk estimates are conservative upper-bound estimates that assume that an individual is exposed according to the RME scenario outlined in this report for all exposure pathways evaluated; and thus represents the maximum possible risk under current land-use conditions.

exceeded unity by a factor of 50. These risk estimates assume that an individual is exposed via all pathways according to the RME case. The highest carcinogenic risk ($1x10^{-3}$) was associated with ingestion of fish from Cobbs Creek. Ingestion of fish and nursing infant exposure pathways had hazard indices that exceeded unity by over an order of magnitude. As presented in Table 6-40, direct contact with surface water in Naylors Run did not significantly increase the risk associated with children playing in this stream.

6.1.5.3 Potential Risks Under Future Land-Use Conditions

Ingestion of Groundwater by Hypothetical Residents - If groundwater at the site were used as a source of water in the future, then residents may be exposed to chemicals of potential concern via ingestion. It is highly unlikely, however, that residents would actually use groundwater in the vicinity of the Havertown PCP site given the availability of municipal water provided by the City of Havertown. This pathway was evaluated primarily to justify further restrictions on groundwater use and provide the basis for making risk management decisions concerning remediation of groundwater at the Havertown PCP site.

Potential carcinogenic risks to hypothetical residents that ingest groundwater from the more contaminated portions of the Havertown PCP site are presented in Table 6-41. Five probable human carcinogens (Group B2) and three known human carcinogens of concern (Group A) were detected groundwater at the site. The primary chemicals of concern in groundwater included benzo(a)pyrene (Equivalent), PCP, and 2,3,7,8-TCDD (Equivalent). The potential increased cancer risk from exposure to these chemicals exceeded 0.1 (the second equation presented in Section 6.1.5.1 was used to estimate carcinogenic risk from these chemicals since the risk level exceeded .01). The total carcinogenic risk for all chemicals was nearly 0.5. The total potential carcinogenic risk associated with ingestion of groundwater was half a million times higher than the NCP point of departure (i.e., $10^{-6}$) and 5,000 times higher than the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). The highest detected

Table 6-41

Potential Carcinogenic Risks Associated with Ingestion of Groundwater from the Havertown PCP Site by Hypothetical Residents for the RME Case

| Chemical | RME Chronic Daily Intake (mg/kg/day) | Slope Factor $(mg/kg/day)^{-1}$ | Weight-of-Evidence | Potential Cancer Risk |
|---|---|---|---|---|
| Organics: | | | | |
| Benzene | 2.8E-3 | 2.9E-2 | A | 8.1E-5 |
| bis(2-Ethylhexyl)phthalate | 2.2E-3 | 1.4E-2 | B2 | 3.1E-5 |
| Benzo(a)pyrene (Equivalent) | 8.9E-3 | 1.2E+1 | B2 | 1.0E-1 |
| Pentachlorophenol | 9.6E-1 | 1.2E-1 | B2 | 1.1E-1 |
| Trichloroethene | 5.6E-3 | 1.1E-2 | B2 | 6.2E-5 |
| Vinyl Chloride | 1.1E-4 | 1.9E+0 | A | 2.1E-4 |
| 2,3,7,8-TCDD (Equivalent) | 2.1E-6 | 1.5E+5 | B2 | 2.7E-1 |
| Inorganics: | | | | |
| Arsenic | 2.7E-4 | 1.7E+0 | A | 4.6E-4 |
| | | | Total Carcinogenic Risk: | 4.9E-1 |

concentrations for these chemicals were found at well locations HAV-02, HAV-04, and R-2 which are installed in the saprolite. Wells installed only in the deep bedrock had significantly lower levels of PCP (factor of 20) and dioxin (factor of 2,000). In addition, carcinogenic PAHs were not detected in the deep bedrock wells. It should be noted, however, that these wells are installed generally along the perimeter of the study area.

Potential noncarcinogenic risks from ingestion of groundwater at the Havertown PCP site are presented in Table 6-42. All of the chemical-specific hazard quotients exceeded unity (1) with the exception of 1,2-dichloroethene (total), bis(2-ethylhexyl)phthalate, arsenic, and thallium. 2,3,7,8-TCDD (Equivalent) and naphthalene had the highest hazard quotients of 5,000 and 170, respectively. The exposure associated with ingestion of 2,3,7,8-TCDD (Equivalent) exceeded the 1-day health advisory by a factor of 50 and the 10-day health advisory by a factor of 500. Thus, ingestion of groundwater at the Havertown PCP site may induce adverse liver effects from acute and subchronic exposure and reproductive effects from chronic exposure.

In addition to the risk estimates calculated according to EPA guidance (1989a), as discussed in Section 6.1.3, risks were estimated for each well location. This was done to provide additional information for making remedial decisions for the site. Essentially, the EPA recommended approach for estimating exposure and risk (presented above) for the groundwater pathway quantifies the risk associated with the hot spot at the site. But such a method does not provide information on the extent and range of risks associated with using groundwater at other locations. Thus, the total risk from ingesting groundwater at each well location was estimated. A contour plot of the carcinogenic and noncarcinogenic risks for the site was then derived using the risks calculated for each well. The risk contour plot defines the spatial distribution of potential carcinogenic risks and noncarcinogenic risks associated with ingestion of groundwater from the Havertown PCP site. A risk contour plot was derived by performing the following analyses.

Table 6-42

Potential Noncarcinogenic Risks Associated with Ingestion of Groundwater from the Havertown PCP site by Hypothetical Residents for the RME Case

| Chemical (a) | RME Chronic Daily Intake (mg/kg/day) | RfD (mg/kg/day) | RfD Uncertainty Factor | Hazard Quotient |
|---|---|---|---|---|
| Organics: | | | | |
| 1,2-Dichloroethene (total) | 7.1E-3 | 2.0E-2 | 1000 | 3.6E-1 |
| bis(2-Ethyhexyl)phthalate | 5.2E-3 | 2.0E-2 | 1000 | 2.6E-1 |
| Fluoranthene | 2.3E-2 | 4.0E-3 | 300 | 5.8E+0 |
| Naphthalene | 6.8E-1 | 4.0E-3 | 10,000 | 1.7E+2 |
| Pentachlorophenol | 2.3E+0 | 3.0E-2 | 100 | 7.7E+1 |
| 2,3,7,8-TCDD (Equivalents)(b) | 5.0E-6 | 1.0E-9 | 1000 | 5.0E+3(b) |
| Inorganics: | | | | |
| Arsenic | 6.5E-4 | 1.0E-3 | 1 | 6.5E-1 |
| Manganese | 6.4E-1 | 1.0E-1 | 1 | 6.4E+0 |
| Thallium | 4.9E-5 | 7.0E-5 | 3000 | 7.0E-1 |
| | | | Total Hazard Index: | 5.3E+3 |

(a) Toxicity criteria were not available for dibenzofuran, 2-methylnaphthalene, acenaphthene, phenanthrene, aluminum, and cobalt; therefore, hazard quotients were not estimated for these chemicals.
(b) Exposure to 2,3,7,8-TCDD (Equivalent) also exceeds both 1- and 10-day health advisories.

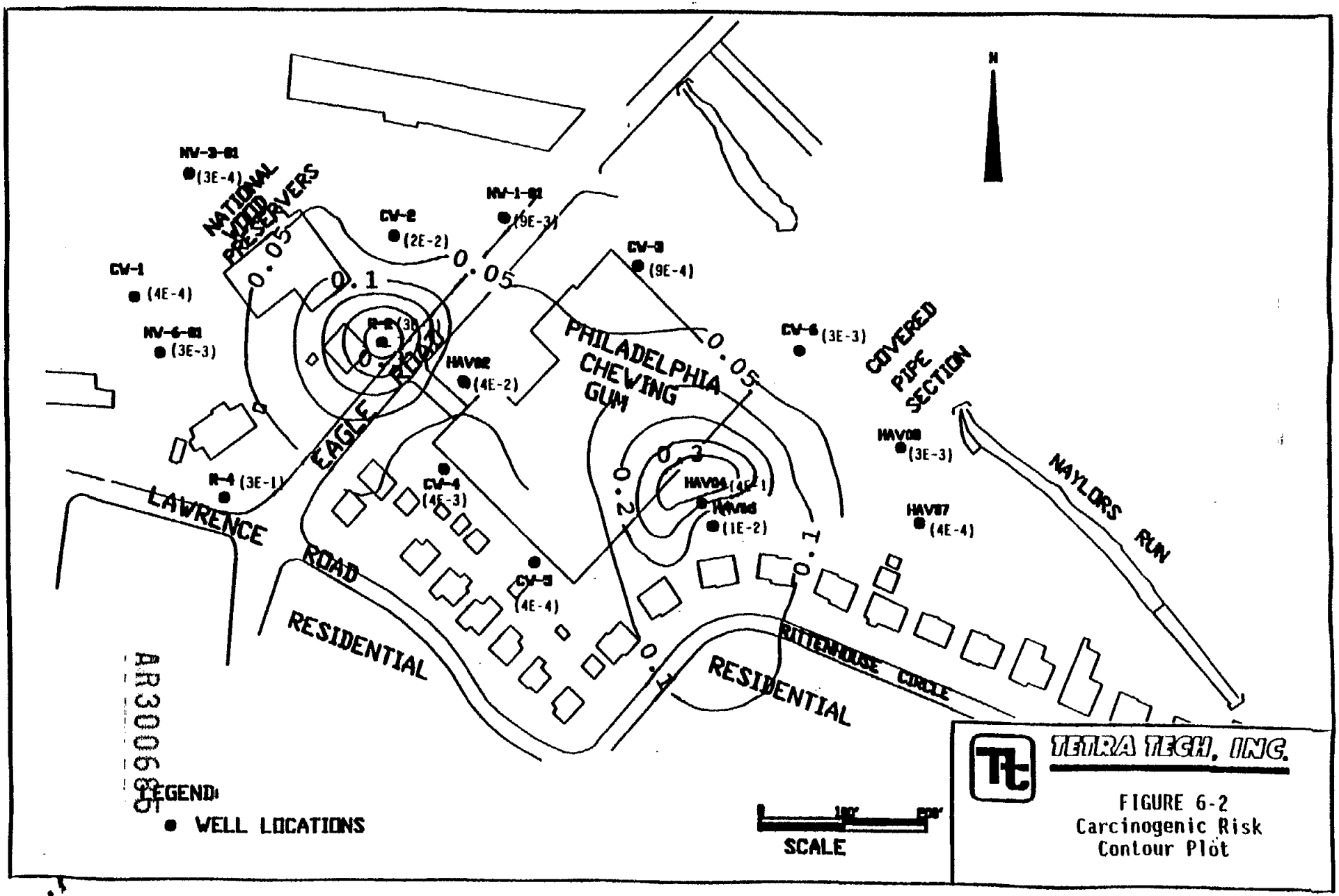
- The potential carcinogenic and noncarcinogenic risk associate with each chemical detected at each well location was estimated using the approaches outlined in Sections 6.1.3.4 and 6.1.5.1.

- The total carcinogenic risk and hazard index for each well location was calculated by summing chemical-specific risks. For well clusters which evaluated contamination in the different portions of the aquifer; the average carcinogenic risk and average hazard index for the location were calculated.

- Carcinogenic risk and noncarcinogenic risk contour plots were derived using a computer contour software system (SURFER Version 4.0).

The risk contour plot for carcinogenic risks associated with ingestion of groundwater at the Havertown PCP site is presented in Figure 6-2. This contour plot shows the major extent of contamination in groundwater as defined by the 0.05 cancer risk contour. The risks associated with groundwater drop off significantly from the 0.05 contour to the risks estimated for wells along the periphery of the study area. As shown in Figure 6-2, potential carcinogenic risks that exceed the NCP acceptable risk range, however, were found at all well locations. The noncarcinogenic risk contour plot presented in Figure 6-3 shows a similar groundwater plume of concern. As shown in Figure 6-3, the hazard index for each well location exceeded unity, with the exception of R-4. These risk contour plots may indicate that contamination of concern with respect to future use of groundwater as a drinking water resource may be found beyond the periphery of the study area (as defined by the current well locations). In addition, these plots indicate the areas of greatest concern with respect to human health.

Inhalation of VOCs while Showering - Potential carcinogenic risks to hypothetical residents who inhale VOCs present in groundwater while showering are presented in Table 6-43. One probable human carcinogen (Group B2) and two known human carcinogens of concern (Group A) which may volatilize while showering were


N
NV-3-01
(3E-4)
NATIONAL WILD RIVERS PRESERVE
NV-1-01
(9E-3)
CV-2
(2E-2)
CV-1
(4E-4)
NV-6-01
(3E-3)
0.05
0.1
0.05
CV-3
(9E-4)
R-2 (3E-2)
EAGLE ROAD
HAV02
(4E-2)
PHILADELPHIA CHEWING GUM
0.05
CV-6 (3E-3)
COVERED PIPE SECTION
HAV08
(3E-3)
NAYLORS RUN
R-1 (3E-1)
LAWRENCE ROAD
CV-4
(4E-3)
0.2
0.2
0.2
1
HAV04 (4E-1)
HAV06
(1E-2)
HAV07
(4E-4)
CV-5
(4E-4)
RESIDENTIAL
RESIDENTIAL
RITTENHOUSE CIRCLE
AR300688
LEGEND:
WELL LOCATIONS
SCALE
TETRA TECH, INC.
FIGURE 6-2
Carcinogenic Risk
Contour Plot

detected groundwater at the site. Trichloroethene is the primary VOC of concern in groundwater. The potential increased cancer risk from exposure to these VOCs while showering is $2x10^{-4}$ which exceeds the NCP point of departure and acceptable risk range (EPA 1990a). The potential carcinogenic risk associated with showering; however, does not contribute significantly to the overall risk of using groundwater as a future drinking water resource. It should be noted; however, that the highest detected concentrations of VOCs were detected in samples further upgradient from the highest detected concentrations of dioxins, PAHs, and PCP.

Potential noncarcinogenic risks from inhalation of VOCs while showering are presented in Table 6-44. 1,2-Dichloroethene (total) was the only VOC with a RfD for evaluating impacts from inhalation. The hazard quotient for 1,2-dichloroethene was below one. Therefore, noncarcinogenic risk from exposure to this chemical may not occur. However, toxicity criteria were not available for evaluating noncarcinogenic effects from inhalation of benzene, trichloroethene, and vinyl chloride.

Indirect Exposure to Nursing Infants from Maternal Exposure to Groundwater - Nursing infants may be indirectly exposed to dioxin and furans in groundwater via lactational transfer assuming that the mother is directly exposed to dioxin and furans via ingestion of groundwater under future land-use conditions. As previously discussed, potential indirect exposure to nursing infants was estimated using a pharmacokinetic model that relates exposure ofthe mother from ingestion of groundwater to the exposure of the nursing infant via lactational transfer. The potential increased carcinogenic risk to nursing infants via indirect exposure is presented in Table 6-38. The increased carcinogenic risk associated with 2,3,7,8-TCDD (Equivalent) exposure to nursing infants (i.e., no additional exposure later in life) was estimated to be $1x10^{-1}$. The potential carcinogenic risk associated with ingestion of groundwater was 10,000 times higher than the NCP point of departure (i.e., $10^{-6}$) and 1,000 times higher than the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$)

Table 6-44

Potential Noncarcinogenic Risks Associated with
Inhalation of VOCs while Showering for Hypothetical
Residents at the Havertown PCP Site for the RME Case

| Chemical (a) | RME Chronic Daily Intake (mg/kg/day) | RfD (b) (mg/kg/day) | RfD Uncertainty Factor | Hazard Quotient |
|---|---|---|---|---|
| 1,2 Dichloroethene (total) | 7.1E-3 | 2.0E-2 | 1000 | 3.6E-1 |

(a) No toxicity criteria were available for benzene, trichloroethene, and vinyl chloride; therefore, the estimated risk does not include these chemicals.

(EPA 1990a).

Potential noncarcinogenic risk to nursing infants via indirect exposure of 2,3,7,8-TCDD (Equivalent) is presented in Table 6-39. The hazard quotient for chronic exposure (i.e., 2 year lactational exposure) exceeded unity by a factor of 39,000. The exposure associated with ingestion of 2,3,7,8-TCDD (Equivalent) exceeded the 1-day health advisory by a factor of 390 and the 10-day health advisory by a factor of 3,900. Thus, indirect exposure to nursing infants which may be indirectly exposed via lactational transfer as a result of maternal exposure under future land-use conditions, may induce adverse liver effects from acute and subchronic exposure and potential developmental effects from chronic exposure. Although, exposure to nursing infants exceeded the 1- and 10-day health advisories, it is still assumed that the exposure duration for the mother from ingestion of groundwater under future land-use conditions is chronic (which would result in significant bioaccumulation of dioxin in the mother prior to lactation).

### 6.1.6 Uncertainties Associated with the Human Health Risk Assessment

This section outlines the uncertainties associated with the results of the Havertown PCP baseline risk assessment. The primary areas of uncertainty include: 1) environmental sampling and analysis; 2) estimation of exposure; and 3) toxicity assessment. An overview of the primary areas of uncertainty in the quantitative risk assessment is presented in Table 6-45 and are discussed below.

#### 6.1.6.1 Environmental Sampling and Analysis

As discussed in Section 6.1.2, monitoring data collected from groundwater, surface water, and sediments were used to characterize the extent of contamination in these media. These data were considered to be representative of site contamination, yet the degree to which the RI data characterizes site contamination is unknown. For example, the potential impact of seasonal

Table 6-45

Uncertainties Associated with the Havertown PCP
Baseline Risk Assessment

| Source of Uncertainty | Effect on Estimated Risk (a) | | |
|---|---|---|---|
| | Potential for Over-Estimation of Risk | Potential for Under-Estimation of Risk | Potential for Over or Under-Estimation of Risk |
| Environmental Sampling and Analysis | | | |
| Available sampling data used to characterize the extent of contamination at the site | | | Low |
| Inorganics were assumed to be elevated above background | Low | | |
| Systematic and/or random errors in analysis and reporting | | | Low |
| TICs were not quantitatively evaluated | | Low | |
| Estimation of Exposure | | | |
| Exposure paramaters were assumed to be characteristic of the potentially exposed population | Moderate | | |
| The amount of media intake is assumed to be constant and representative of the exposed population | Moderate | | |
| Toxicity Assessment | | | |
| An additive model is used to evaluate risk from a chemical mixture | | | Moderate |
| Toxicity criteria not available for certain chemicals of potential concern | | Low | |
| Conservative methods used to derive toxicity criteria (particularly slope factors [see text]) | Moderate to high | | |

(a)As a general guideline, assumptions marked as "low," may affect estimates of exposure by less than one order of magnitude; assumptions marked "moderate" may affect estimates of exposure by between one and two orders of magnitude; and assumptions marked "high" may affect estimates of exposure by more than two orders of magnitude.

AR300691

variability on site contamination was not characterized since this was not within the scope of the RI. Given the uncertainty associated with the monitoring data, the 95th UCL on the arithmetic mean was used when estimating exposure for the various exposure pathways evaluated in this assessment in order that potential exposure would not be underestimated.

Another area of uncertainty concerns the treatment of non-detected concentrations in the quantitative assessment of risk. One-half of the CRQL was used as the detection limit for samples qualified with a "U" or "UJ" qualifier. The actual concentration of the chemical may be zero to just below the CRQL. In all probability, the actual concentration may be below one-half the CRQL given that the instrument detection limit (IDL) is often much lower than one-half the CRQL. The methods used to evaluate non-detects in this assessment, however, probably does not contribute significantly to the overall uncertainty of the results (probably less than a factor of 2).

In this assessment, several inorganic chemicals of potential concern were selected for evaluation in the quantitative risk assessment as discussed in Section 6.1.2. Site-specific background data, however, were not available for groundwater, surface water, or sediment. Monitoring wells installed upgradient from the suspected source areas had significant organic contamination and; therefore, could not be considered as background wells for groundwater. The site is located at the headwaters of Naylors Run; therefore, site specific background data could not be collected. To be conservative, inorganic chemicals detected in groundwater, surface water, and sediment which are not essential human nutrients and contributed significantly to overall risk (i.e., greater than 1 percent of carcinogenic and/or noncarcinogenic risk) were assumed to be elevated above background concentrations. Thus, these inorganic chemicals were selected as chemicals of potential concern. The risks presented in this report would be overestimated if any or all of the inorganic chemicals are attributable to background levels. It should be noted, however, that inorganic chemicals were not the primary chemicals of concern at the site and thus would not significantly

impact the results of the baseline risk assessment.

Another potential source of uncertainty involves the analytical methods used to quantify the levels of chemicals of potential concern in samples collected for the Havertown PCP site. There is a certain degree of variability associated with the laboratory instruments ability to quantify the levels of a chemical in a sample. This variability tends to be normally distributed. The potential contribution of this source of uncertainty, however, is considered to be low given QA/QC requirements for samples and analysis.

Several TICs were identified in groundwater, surface water, and sediment. Given the uncertainty associated with their identification and concentrations, these chemicals were not quantitatively evaluated in this report. Thus, the risks associated with contact with various media may be underestimated. Alkyl benzene, PAHs, and breakdown products of PCP were the primary TICs identified.

6.1.6.2 Estimation of Exposure

As discussed in Sections 6.1.3 and 6.1.5, conservative assumptions were used to estimate exposure for the various exposure pathways quantitatively evaluated in this report. Under current land-use conditions, it was assumed that children would play in the more contaminated areas of the Naylors Run 125 days per year for 10 years. During these play activities, children would incidentally ingest 140 mg of sediment each day. In addition, children were assumed to contact surface water and sediments over one-third of the surface area of their hands, arms, and legs. These are conservative assumptions used to evaluate a reasonable maximum exposure case. The likelihood of children in the area actually engaging in such behavior is unknown.

For the fish ingestion pathway, recreational fisherman were assumed to ingest an average of 42 grams per day of bottom feeding fish from Cobbs Creek. No data were available for game fish which are more likely to be ingested by recreational

fisherman. Game fish may have much lower concentrations of organic contaminants in their tissue than bottom feeding fish given the differences in their foraging behavior. Therefore, potential exposure levels may be overestimated.

For future land-use exposure pathways, it was assumed that an individual would ingest 2 liters per day of groundwater from more contaminated areas at the site over a 30 year period. It is unlikely that groundwater at the site would actually be used as a future drinking water resource. This pathways, however, was evaluated primarily to justify restrictions on the future use of groundwater at the site and provide the basis for making risk management decisions for the site.

6.1.6.3 Toxicity Assessment

EPA (1989a, 1986a,b) recommends summing chemical-specific risks in order to quantify the combined risk associated with exposure to a chemical mixture. Limited data are available for actually quantifying the potential synergistic and/or antagonistic relationships between chemicals in a chemical mixture. Thus, chemicals are assumed to act independently in the body to cause an effect. If this assumption is incorrect regarding chemical interaction, then over- or underestimation of potential risk of the chemical mixture may occur.

Several chemicals of potential concern, presented in Section 6.1.2, did not have available toxicity criteria. Therefore, the potential noncarcinogenic and carcinogenic risks associated with the site may be underestimated. However, the chemicals of primary concern at the Havertown PCP site have available toxicity criteria. Therefore, the uncertainty associated with the lack of toxicity criteria for other chemicals of potential concern is considered low.

There is a high degree of uncertainty associated with the derivation of available toxicity criteria. The primary sources of uncertainty associated with the derivation of toxicity criteria, as summarized by the EPA (1989a), include:

- using dose-response information from effects observed at high doses to predict the adverse health effects that may occur following exposure to the low levels expected from human contact with the agent in the environment;

- using dose-response information from short-term exposure studies to predict the effects of long-term exposures, and vice-versa;

- using dose-response information from animal studies to predict effects in humans; and

- using dose-response information from homogeneous animal populations or healthy human populations to predict the effects likely to be observed in the general population consisting of individuals with a wide range of sensitivity.

EPA (1989a,e,f, 1986a,b) uses a conservative approach to derive toxicity criteria given the uncertainties in the toxicity studies and dose-response information. For example, the slope factor is the 95th UCL on the linear slope that describes the cancer potency of the chemical of concern. Using the 95th UCL on the linear slope is a conservative approach adopted by the EPA in order that the true risks will not be underestimated. A thorough assessment of the high degree of uncertainty associated with the derivation of slope factors was presented in an EPA (1985e) document entitled "Techniques for the Assessment of the Carcinogenic Risk to the U.S. Population Due to Exposure from Selected Volatile Organic Compounds from Drinking Water Via the Ingestion, Inhalation, and Dermal Routes." Based on the conservative approaches used to derive slope factors outlined in this report (EPA 1985e), it may be concluded that the "true carcinogenic risk" may be orders of magnitude less than the carcinogenic risks presented in this report.

Thus, risks presented in the Havertown PCP baseline risk assessment should not

be construed as absolute estimates of risk given the degree of uncertainty associated with the risk assessment process as described above. Rather, the Havertown PCP baseline risk assessment characterizes the potential for an adverse effect to occur if an individual is exposed to chemicals of concern at the site. When reviewing the results of this assessment, the conservative assumptions used should be considered. The conservative methods are recommended in EPA guidance (1989a) in order to ensure that risks are not underestimated.

### 6.1.7 Summary and Conclusions of the Human Health Risk Assessment

This section summarizes the findings of the human health risk assessment for the Havertown PCP site. This report determines whether chemicals of potential concern at the Havertown PCP site pose a current or future risk to human health under the no-action alternative (i.e., in the absence of remediation of the site). Chemicals of potential concern selected for evaluation in the baseline risk assessment are discussed in Section 6.1.7.1. Exposure pathways of concern selected for quantitative evaluation in the baseline risk assessment are summarized in Section 6.1.7.2. Potential carcinogenic and noncarcinogenic risks estimated for the pathways quantitatively evaluated in this report are summarized below in Section 6.1.7.3

#### 6.1.7.1 Chemicals of Potential Concern

Of the chemicals detected at the Havertown PCP site, chemicals of potential concern were selected based on several criteria including evaluating the percent contribution of risk using derived risk factors (EPA 1989a). Over forty chemicals were selected as chemicals of potential concern for the Havertown PCP site including volatile organic compounds, PAHs, pesticides, dioxins and furans, and inorganics. Of these chemicals, PCP, PAHs (specifically benzo(a)pyrene [Equivalents]), dioxins and furans were the primary chemicals of concern in all media at the Havertown PCP site. Other chemicals selected as chemicals of

potential concern in all media included: aluminum, arsenic, cobalt, and manganese. Several volatile organic compounds selected as chemicals of potential concern were detected only in groundwater including: 1,2-dichloroethene, trichloroethene, and vinyl chloride. The exclusive presence of these chemicals in groundwater may be due to their high water solubility, low affinity for binding to sediment particles, and potential volatilization from surface water to the air. The pesticides dieldrin and heptachlor epoxide were only detected in Naylors Run surface water. The majority of the PAHs were found in sediment samples, probably due to their low water solubility and high affinity for binding to sediment particles. Several inorganic chemicals of potential concern including antimony, nickel, thallium, vanadium, and zinc were selected only in Naylors Run. It is uncertain whether these chemicals are actually associated with site related disposal. TICs identified in groundwater and surface water consisted primarily of alkyl benzenes and PAHs. The TICs identified in sediment consisted of PAHs and breakdown products of PCP. The presence of these TICs is consistent with the disposal history of the site.

6.1.7.2 Exposure Assessment

The following current land-use exposure pathways were quantitatively evaluated in the Havertown PCP baseline risk assessment report:

- direct contact with surface water and sediments by children playing in Naylors Run;
- ingestion of fish caught from Cobbs Creek by recreational fisherman; and
- exposure to nursing infants that ingest breast-milk from mothers that are exposed to dioxin via ingestion of fish from Cobbs Creek.

The following future land-use exposure pathways were quantitatively evaluated in

the Havertown PCP baseline risk assessment report:

- ingestion of groundwater at the Havertown PCP site by future hypothetical residents;

- inhalation of volatile organic compounds (VOCs) while showering by future hypothetical residents that use groundwater at the Havertown PCP site; and

- exposure to nursing infants that ingest breast-milk from mothers that are exposed to dioxin via ingestion of groundwater.

Exposure point concentrations were estimated for each chemical of potential concern and exposure pathway. Exposure point concentrations and exposure parameters values were combined using a chemical intake equation to estimate exposure (i.e., chronic daily intake [CDI]) for the reasonable maximum exposure (RME) case for each chemical of potential concern and pathway.

6.1.7.3 Results of the Human Health Risk Characterization

Toxicity criteria identified in Section 6.1.4 and CDIs estimated in Section 6.1.3 were combined to quantify potential noncarcinogenic and carcinogenic risks associated with the exposure pathways quantitatively evaluated in the Havertown PCP baseline risk assessment.

Potential carcinogenic risk was quantified by multiplying the CDI by the slope factor when the cancer risk was below 0.01. Cancer risks in excess of 0.01, were calculated using an inverse exponential equation presented in Section 6.1.5.1. Chemical-specific cancer risks were summed in order to quantify the total cancer risk associated with exposure to a chemical mixture. Potential carcinogenic risks are expressed as an increased probability of developing cancer over a lifetime (i.e., excess individual lifetime cancer risk) (EPA 1989a). For

example, a $10^{-6}$ increased cancer risk can be interpreted as an increased risk of 1 in 1,000,000 for developing cancer over a lifetime if an individual is exposed as defined by the pathways presented in this report. A $10^{-6}$ increased cancer risk is the point of departure established in the NCP (EPA 1990a). In addition, the NCP (EPA 1990a) states that "for known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between $10^{-4}$ and $10^{-6}$." Carcinogenic risks in excess of the acceptable risk range are likely to trigger a remedial response. Carcinogenic risks within the acceptable risk range, yet in excess of the point of departure (i.e., $10^{-6}$), also may trigger a remedial response.

Noncarcinogenic effects associated with exposure to a chemical was quantified by dividing its CDI with its reference dose (RfD). This ratio is called the hazard quotient. If the hazard quotient exceeds unity (i.e., 1), then an adverse health effect may occur. If the estimated hazard quotient is less than unity, then adverse noncarcinogenic effects are unlikely to occur. The potential risk from a chemical mixture was evaluated by calculating the hazard index which is the sum of the chemical-specific hazard quotients.

As discussed in Section 6.1.3.3, Section 6.1.5, and Section 6.1.7, conservative assumptions were used to estimate CDIs and risk in order that potential risk will not be underestimated. The conservative assumptions are used because of the uncertainty associated with the risk assessment process. The assumptions discussed in this report should be considered when reviewing the risks presented in this section. In particular, the risk estimates presented for future use of groundwater should be interpreted as an evaluation of groundwater quality at the site for developing remediation strategies. Groundwater in the vicinity of the Havertown PCP site is currently not used as a drinking water resource. In addition, it is highly unlikely that groundwater would be used as a drinking water resource in the future given the availability of city water provided by the

City of Havertown.

A summary of the potential carcinogenic and noncarcinogenic risks estimated for the exposure pathways quantitatively evaluated in the Havertown PCP baseline risk assessment are presented in Table 6-46 and discussed below.

Current Land-Use Conditions: Direct Contact with Surface Water by Children Playing in Naylors Run - The total carcinogenic risk to children playing in Naylors Run from dermal absorption of chemicals of potential concern in surface water is $9x10^{-7}$ for the RME case. The potential carcinogenic risk associated with direct contact with surface was below the point of departure established in the NCP (EPA 1990a). All of the chemical-specific hazard quotients were nearly 3 orders of magnitude below unity (1) for the RME case. In addition, the hazard index was nearly 2 orders of magnitude below unity for the RME case. Thus, surface water in Naylors Run does not appear to present an appreciable carcinogenic risk nor noncarcinogenic risk to children who may play in this stream, given the estimated risk levels and the conservative assumptions used to assess exposure (e.g., high frequency of exposure, playing exclusively in the most contaminated area at the site, etc.).

Current Land-Use Conditions: Direct Contact with Sediments by Children Playing in Naylors Run - The total carcinogenic risk to children playing in Naylors Run from dermal absorption of chemicals of potential concern in sediment was $6x10^{-5}$ for the RME case. The majority of the carcinogenic risk was associated with benzo(a)pyrene (Equivalent) and arsenic. The total carcinogenic risk to children playing in Naylors Run from incidental ingestion of chemicals of potential concern in sediment was $5x10^{-5}$ for the RME case. The majority of the carcinogenic risk for this route was associated with benzo(a)pyrene (Equivalent) (the dermal absorption of arsenic was assumed to be negligible). The highest detected concentrations of benzo(a)pyrene (Equivalents) and arsenic were found

Table 6-46
Conclusions of the Havertown PCP
Baseline Risk Assessment

| Exposure Pathway | Potential Carcinogenic Risk | Potential Noncarcinogenic Risk (Hazard Index)(HI) | Comments |
|---|---|---|---|
| Current Land-Use Conditions | | | |
| Children Playing in Naylors Run | 1E-4 | <1(0.7) | Relatively low risk from direct contact with surface water. Majority of the potential carcinogenic risk associated with Benzo(a)pyrene (Equivalent) and arsenic in sediments. Highest levels of benzo(a)pyrene (Equivalent) and arsenic were found upstream of the catch basin. HI slightly below unity; therefore, noncarcinogenic risk may not occur. |
| Ingestion of Fish from Cobbs Creek | 2E-3 | 13.6 | Carcinogenic risk exceeds the upper-bound of NCP acceptable risk range (i.e., $10^{-4}$). Majority of carcinogenic risk from dieldrin which was detected in surface water at Naylors Run. HI exceeds unity; therefore, recreational fishermen that ingest significant quantities of fish from Cobbs Creek may experience noncarcinogenic effects. Chlordane, dieldrin, heptachlor epoxide, and dioxin in fish all contributed significantly to risk. Unclear whether these chemicals are associated with the site. |
| Indirect Exposure to Nursing Infants (Maternal exposure from ingestion of fish) | 1E-4 | 33 | Increased carcinogenic risk from dioxin equals the upper-bound of the NCP acceptable risk range (i.e., $10^{-4}$). Hazard quotient for dioxin exceeds unity for chronic exposure and 10-day health advisory. Therefore, nursing infants may experience adverse liver and developmental effects. |

Table 6-46(Cont.)
Conclusions of the Havertown PCP
Baseline Risk Assessment

| Exposure Pathway | Potential Carcinogenic Risk | Potential Noncarcinogenic Risk (Hazard Index)(HI) | Comments |
|---|---|---|---|
| Future Land-Use Conditions | | | |
| Ingestion of Groundwater by Hypothetical Resident | 5E-1 | 5E+3 | Carcinogenic risk is 1,000,000 times higher than the the NCP point of departure (i.e., $10^{-6}$) and 10,000 times higher than the upper-bound of the NCP acceptable risk range. The majority of the carcinogenic risk associated with benzo(a)pyrene (equivalent), PCP, and dioxin (See Figure 6-2 for spatial distribution of cancer risk). HI exceeds unity by a factor of 5000 (reproductive effects). The majority of the noncarcinogenic risk associated with dioxin. Exposure to dioxin also exceeds 1-day and 10-day health advisories (adverse liver effects). |
| Inhalation of VOCS in Groundwater by Hypothetical Residents while Showering | 4E-2 | <1(0.4) | Carcinogenic risk from benzene, TCE, and vinyl chloride exceeds upper-bound of the NCP acceptable risk range (i.e.,$10^{-4}$). The risk from showering, however, does not contribute significantly to risk from ingestion. Highest levels of VOCs in groundwater upgradient from PAH, PCP, and dioxin "hot spots." HI slightly exceeds unity, due to exposure to TCE. |
| Indirect Exposure to Nursing infants (Maternal exposure from ingestion of groundwater) | 1E-1 | 4E+4 | Increased carcinogenic risk from dioxin exceeds the upper-bound of the NCP acceptable risk range (i.e., (i.e., $10^{-4}$) by a factor of 1,000. Hazard quotient for dioxin exceeds unity by a factor of 39,000; as well as 1-day and 10-day health advisories by factors of 390 and 3,900 respectively. Therefore, nursing infants may experience developmental problems from chronic exposure and liver problems from subchronic and acute exposure. |

upstream of the catch basin in samples collected in Naylors Run near Eagle Road. The total potential carcinogenic risk to children from contact with sediments was $1x10^{-4}$ for the RME case. This estimated cancer risk is above the NCP point of departure (i.e., $10^{-6}$) and equal to the upper-bound of the NCP acceptable risk range (i.e., $10^{-4}$) (EPA 1990a). It should be noted, however, that conservative methods were used to estimate exposure to children playing in Naylors Run (e.g., high frequency of exposure, playing exclusively in the most contaminated area at the site, etc.).

For this pathway, all of the chemical-specific hazard quotients were below unity (1) and the total hazard index for exposure to sediment was 0.9 for the RME case. Therefore, noncarcinogenic effects may not occur in children from dermal absorption and incidental ingestion of chemicals of potential concern in sediment during playing activities.

The potential noncarcinogenic risk associated with exposure to lead in sediments was evaluated using the Integrated Uptake/Biokinetic Model (IU/BK) which is a computerized pharmacokinetic model. Lead was not a chemical of concern in any other media; therefore, default parameter values were used to estimate exposure to lead from other media (i.e., drinking water, air, etc.). Based on the results of the IU/BK model, there is a 9 percent chance that a child engaged in the activity outlined for this pathway would have a blood-lead level above 10 µg/dl. Studies have shown that children with blood-lead levels above 10 µg/dl may experience adverse neurological effects (see toxicity profile for lead for further discussion).

Current Land-Use Conditions: Ingestion of Fish from Cobbs Creek - Fish tissue samples collected as part of the National Bioaccumulation Study from Cobbs Creek were used to estimate potential exposure to recreational fisherman (EPA 1990d). The total carcinogenic risk associated with ingestion of fish tissue was $2x10^{-3}$ for the RME case. The majority of the carcinogenic risk was associated with

dieldrin. Dieldrin was detected in Naylors Run surface water, but not in any other media. It is uncertain whether dieldrin or other chemicals present in fish tissue are associated with chemical releases from the Havertown PCP site or other sources. The total potential carcinogenic risk associated with ingestion of fish tissue is above the NCP point of departure (i.e., $10^{-6}$) and the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). The hazard index for ingestion of fish was 14 for the RME case. Hazard quotients for chlordane, dieldrin, heptachlor epoxide, and dioxin all exceeded unity (1) for the RME case. Therefore, ingestion of large quantities of bottom feeding fish (no game fish data were available) from Cobbs Creek may result in a noncarcinogenic effect.

Current Land-Use Conditions: Indirect Exposure to Nursing Infants from Maternal Exposure to Fish - Nursing infants may be indirectly exposed to dioxin and furans in fish tissue via lactational transfer assuming that the mother is directly exposed to dioxin and furans in fish tissue from Cobbs Creek. Potential exposure to nursing infants was estimated using a pharmacokinetic model that relates exposure of the mother from ingestion of fish to the exposure of the nursing infant via lactational transfer. The increased carcinogenic risk associated with 2,3,7,8-TCDD (Equivalent) exposure for nursing infants (i.e., no additional exposure later in life) was estimated to be $1x10^{-4}$ for the RME case. The increased risk is above the NCP point of departure (i.e., $10^{-6}$) and is equal to the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). The hazard quotient for chronic exposure (i.e., 2 year lactational exposure) exceeded unity by an order of magnitude for the RME case. Therefore, nursing infants may experience adverse developmental effects from chronic exposure.

Current Land-Use Conditions: Multimedia Assessment of Risk - The total carcinogenic risk associated with exposure to all pathways under current land-use conditions was $2x10^{-3}$, while the hazard quotient exceeded unity by a factor of

50 for the RME case. These risk estimates assume that an individual is exposed via all pathways according to the RME case. The highest carcinogenic risk ($1x10^{-3}$) was associated with ingestion of fish from Cobbs Creek. Ingestion of fish and nursing infant exposure pathways had hazard indices that exceeded unity by over an order of magnitude.

Future Land-Use Conditions: Ingestion of Groundwater by Hypothetical Residents - If groundwater at the site were used as a source of water in the future, then residents may be exposed to chemicals of potential concern via ingestion. The total carcinogenic risk for all chemicals was nearly 0.5 for the RME case. The total potential carcinogenic risk associated with ingestion of groundwater was one-half a million times higher than the NCP point of departure (i.e., $10^{-6}$) and 5,000 times higher than the upper-bound of the acceptable risk range as presented in the NCP (i.e., $10^{-4}$) (EPA 1990a). The primary chemicals of concern in groundwater included benzo(a)pyrene (Equivalent), PCP, and 2,3,7,8-TCDD (Equivalent). The highest detected concentrations of these chemicals were found at well locations HAV-02, HAV-04, and R-2 (see risk contour plot presented in Figure 6-2 for delineation of the plume for carcinogenic risk). Deep bedrock wells which are generally installed along the perimeter of the study area, however, had significantly lower concentrations of these chemicals.

The hazard index estimated for ingestion of groundwater exceeded unit by a factor of over 5,000 for the RME case. Over 95 percent of the noncarcinogenic risk was associated with 2,3,7,8-TCDD (Equivalent). Exposure associated with ingestion of 2,3,7,8-TCDD (Equivalent) exceeded the 1-day health advisory by a factor of 50 and the 10-day health advisory by a factor of 500. Thus, ingestion of groundwater at the Havertown PCP site may induce adverse liver effects from acute and subchronic exposure and reproductive effects from chronic exposure (see risk contour plot presented in Figure 6-3 for delineation of the plume for noncarcinogenic risk).

Future Land-Use Conditions: Inhalation of VOCs while Showering - The potential increased cancer risk from exposure to VOCs in groundwater while showering is $2x10^{-4}$ for the RME case, which exceeds the NCP point of departure and acceptable risk range (EPA 1990a). It is uncertain whether VOCs in groundwater may cause a noncarcinogenic effect from inhalation given the lack of toxicity criteria for benzene, trichloroethene, and vinyl chloride.

Indirect Exposure to Nursing Infants from Maternal Exposure to Groundwater - Nursing infants may be indirectly exposed to dioxin and furans in groundwater via lactational transfer assuming that the mother is directly exposed to dioxin and furans via ingestion of groundwater under future land-use conditions. The increased carcinogenic risk associated with 2,3,7,8-TCDD (Equivalent) exposure to nursing infants (i.e., no additional exposure later in life) was estimated to be $1x10^{-1}$ for the RME case. The potential carcinogenic risk associated with ingestion of groundwater was 10,000 times higher than the NCP point of departure (i.e., $10^{-6}$) and 1,000 times higher than the upper-bound of the acceptable risk range, as presented in the NCP (i.e., $10^{-4}$) EPA (1990a).

The hazard quotient for chronic exposure (i.e., 2 year lactational exposure) exceeded unity by a factor of 39,000 for the RME case. The exposure associated with ingestion of 2,3,7,8-TCDD (Equivalent) exceeded the 1-day health advisory by a factor of 390 and the 10-day health advisory by a factor of 3,900. Thus, indirect exposure to nursing infants via lactational transfer as a result of maternal exposure under future land-use conditions, may induce adverse liver effects from acute and subchronic exposure and potential developmental effects from chronic exposure.

Overall, the primary conclusions of the Havertown PCP baseline risk assessment are as follows:

- Carcinogenic PAH contamination in sediments may present a potential human health impact from direct contact. Pesticides and dioxin in surface water and sediments may contribute to the health risk associated with ingestion of fish further downstream and subsequent indirect exposure to nursing infants. However, it is uncertain whether these chemicals present in fish tissue are associated with chemicals releases from the site

- There are high carcinogenic and noncarcinogenic risks associated with the use of groundwater due to PAH, PCP, and dioxin contamination. The extent of primary contamination of these chemicals appears to be sufficiently characterized by data from existing monitoring wells. However, relatively low concentrations of these chemicals in monitoring wells installed along the periphery of the study area may present risks of concern with respect to residential use of groundwater.